TOXICOLOGICAL PROFILE FOR

TOTAL PETROLEUM HYDROCARBONS (TPH)

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

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UPDATE STATEMENT

A Toxicological Profile for Total Petroleum Hydrocarbons (TPH) was released in September 1998. This edition supersedes any previously released draft or final profile.

Toxicological profiles are revised and republished as necessary, but no less than once every three years. For information regarding the update status of previously released profiles, contact ATSDR at:

Agency for Toxic Substances and Disease Registry
Division of Toxicology/Toxicology Information Branch
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333

FOREWORD

This toxicological profile is prepared in accordance with guidelines developed by the Agency for Toxic Substances and Disease Registry (ATSDR) and the Environmental Protection Agency (EPA). The original guidelines were published in the *Federal Register* on April 17, 1987. Each profile will be revised and republished as necessary.

The ATSDR toxicological profile succinctly characterizes the toxicologic and adverse health effects information for the hazardous substance described therein. Each peer-reviewed profile identifies and reviews the key literature that describes a hazardous substance's toxicologic properties. Other pertinent literature is also presented, but is described in less detail than the key studies. The profile is not intended to be an exhaustive document; however, more comprehensive sources of specialty information are referenced.

The focus of the profiles is on health and toxicologic information; therefore, each toxicological profile begins with a public health statement that describes, in nontechnical language, a substance's relevant toxicological properties. Following the public health statement is information concerning levels of significant human exposure and, where known, significant health effects. The adequacy of information to determine a substance's health effects is described in a health effects summary. Data needs that are of significance to protection of public health are identified by ATSDR and EPA.

Each profile includes the following:

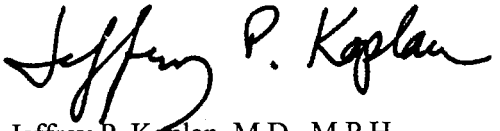
(A) The examination, summary, and interpretation of available toxicologic information and epidemiologic evaluations on a hazardous substance to ascertain the levels of significant human exposure for the substance and the associated acute, subacute, and chronic health effects;

(B) A determination of whether adequate information on the health effects of each substance is available or in the process of development to determine levels of exposure that present a significant risk to human health of acute, subacute, and chronic health effects; and

(C) Where appropriate, identification of toxicologic testing needed to identify the types or levels of exposure that may present significant risk of adverse health effects in humans.

The principal audiences for the toxicological profiles are health professionals at the Federal, State, and local levels; interested private sector organizations and groups; and members of the public.

This profile reflects ATSDR's assessment of all relevant toxicologic testing and information that has been peer-reviewed. Staff of the Centers for Disease Control and Prevention and other Federal scientists have also reviewed the profile. In addition, this profile has been peer-reviewed by a nongovernmental panel and is being made available for public review. Final responsibility for the contents and views expressed in this toxicological profile resides with ATSDR.



Jeffrey P. Koplan, M.D., M.P.H.
Administrator
Agency for Toxic Substances and
Disease Registry

*Legislative Background

The toxicological profiles are developed in response to the Superfund Amendments and Reauthorization Act (SARA) of 1986 (Public Law 99-499) which amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund). Section 211 of SARA also amended Title 10 of the U. S. Code, creating the Defense Environmental Restoration Program. Section 2704(a) of Title 10 of the U. S. Code directs the Secretary of Defense to notify the Secretary of Health and Human Services of not less than 25 of the most commonly found unregulated hazardous substances at defense facilities. Section 2704(b) of Title 10 of the U. S. Code directs the Administrator of the Agency for Toxic Substances and Disease Registry (ATSDR) to prepare a toxicological profile for each substance on the list provided by the Secretary of Defense under subsection (b).

CONTRIBUTORS

CHEMICAL MANAGER(S)/AUTHORS(S):

G. Daniel Todd, Ph.D.
ATSDR, Division of Toxicology, Atlanta, GA

Robert L. Chessin, M.S.P.H.
Research Triangle Institute, Research Triangle Park, NC

Joan Colman, Ph.D.
Syracuse Research Corporation, Syracuse, NY

THE PROFILE HAS UNDERGONE THE FOLLOWING ATSDR INTERNAL REVIEWS:

1. Health Effects Review. The Health Effects Review Committee examines the health effects chapter of each profile for consistency and accuracy in interpreting health effects and classifying end points.

3. Minimal Risk Level Review. The Minimal Risk Level Workgroup considers issues relevant to substance-specific minimal risk levels (MRLs), reviews the health effects database of each profile, and makes recommendations for derivation of MRLs.

4. Science Policy Oversight Committee. The SPOC reviews documents for consistency with ATSDR policy.

4. Data Needs Review. The Research Implementation Branch reviews data needs sections to assure consistency across profiles and adherence to instructions in the Guidance.

PEER REVIEW

A peer review panel was assembled for total petroleum hydrocarbons. The panel consisted of the following members:

1. Barbara Callahan, Ph.D., Director of Risk Assessment Services, Fluor Daniel GTI, Norwood, Massachusetts;

2. Michael Hutcheson, Ph.D, MPH, Deputy Director, Air and Water Toxics, Office of Research and Standards, Massachusetts Department of Environmental Protection, Boston, Massachusetts; and

3. Paul Kostecki, Ph.D., Professor, University of Massachusetts, School of Public Health, Amherst, Massachusetts.

These experts collectively have knowledge of total petroleum hydrocarbons' physical and chemical properties, toxicokinetics, key health end points, mechanisms of action, human and animal exposure, and quantification of risk to humans. All reviewers were selected in conformity with the conditions for peer review specified in Section 104(i)(13) of the Comprehensive Environmental Response, Compensation, and Liability Act, as amended.

Scientists from the Agency for Toxic Substances and Disease Registry (ATSDR) have reviewed the peer reviewers' comments and determined which comments will be included in the profile. A listing of the peer reviewers' comments not incorporated in the profile, with a brief explanation of the rationale for their exclusion, exists as part of the administrative record for this compound. A list of databases reviewed and a list of unpublished documents cited are also included in the administrative record.

The citation of the peer review panel should not be understood to imply its approval of the profile's final content. The responsibility for the content of this profile lies with the ATSDR.

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1. PUBLIC HEALTH STATEMENT

This public health statement tells you about total petroleum hydrocarbons (TPH) and the effects of exposure. The Environmental Protection Agency (EPA) identifies the most serious hazardous waste sites in the nation. These sites make up the National Priorities List (NPL) and are the sites targeted for long-term federal cleanup activities. TPH, itself, has been reported at 34 of the 1,519 current or former NPL sites. Many NPL sites are contaminated with components of TPH, though no estimate has been made of the number of these sites. This information is important because exposure to these components may harm you and because these sites may be sources of exposure.

When a substance is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment. This release does not always lead to exposure. You are exposed to a substance only when you come in contact with it. You may be exposed by breathing, eating, or drinking the substance or by skin contact.

If you are exposed to TPH, many factors determine whether you'll be harmed. These factors include the dose (how much), the duration (how long), and how you come in contact with it. You must also consider the other chemicals you're exposed to and your age, sex, diet, family traits, lifestyle, and state of health.

1.1 WHAT ARE TOTAL PETROLEUM HYDROCARBONS?

Total Petroleum Hydrocarbons (TPH) is a term used to describe a broad family of several hundred chemical compounds that originally come from crude oil. In this sense, TPH is really a mixture of chemicals. They are called hydrocarbons because almost all of them are made entirely from hydrogen and carbon. Crude oils can vary in how much of each chemical they contain, and so can the petroleum products that are made from crude oils. Most products that contain TPH will bum. Some are clear or light-colored liquids that evaporate easily, and others are thick, dark

liquids or semi-solids that do not evaporate. Many of these products have characteristic gasoline, kerosene, or oily odors. Because modern society uses so many petroleum-based products (for example, gasoline, kerosene, fuel oil, mineral oil, and asphalt), contamination of the environment by them is potentially widespread. Contamination caused by petroleum products will contain a variety of these hydrocarbons. Because there are so many, it is not usually practical to measure each one individually. However, it is useful to measure the total amount of all hydrocarbons found together in a particular sample of soil, water, or air.

The amount of TPH found in a sample is useful as a general indicator of petroleum contamination at that site. However, this TPH measurement or number tells us little about how the particular petroleum hydrocarbons in the sample may affect people, animals, and plants. By dividing TPH into groups of petroleum hydrocarbons that act alike in the soil or water, scientists can better know what happens to them. These groups are called petroleum hydrocarbon fractions. Each fraction contains many individual compounds. Much of the information in this profile talks about TPH fractions. See Chapter 2 for more information on what components make up TPH and how they are measured.

1.2 WHAT HAPPENS TO TPH WHEN IT ENTERS THE ENVIRONMENT?

TPH is released to the environment through accidents, as releases from industries, or as byproducts from commercial or private uses. When TPH is released directly to water through spills or leaks, certain TPH fractions will float in water and form thin surface films. Other heavier fractions will accumulate in the sediment at the bottom of the water, which may affect bottom- feeding fish and organisms. Some organisms found in the water (primarily bacteria and fungi) may break down some of the TPH fractions. TPH released to the soil may move through the soil to the groundwater. Individual compounds may then separate from the original mixture, depending on the chemical properties of the compound. Some of these compounds will evaporate into the air and others will dissolve into the groundwater and move away from the release area. Other compounds will attach to particles in the soil and may stay in the soil for a long period of

time, while others will be broken down by organisms found in the soil. See Chapter 5 for more information on how TPH enters and spreads through the environment.

1.3 HOW MIGHT I BE EXPOSED TO TPH?

Everyone is exposed to TPH from many sources, including gasoline fumes at the pump, spilled crankcase oil on pavement, chemicals used at home or work, or certain pesticides that contain TPH components as solvents. A small amount of lighter TPH components are found in the general air you breathe. Many occupations involve extracting and refining crude oil, manufacturing petroleum and other hydrocarbon products, or using these products. If you work with petroleum products, you may be exposed to higher levels of TPH through skin contact or by breathing contaminated air. If TPH has leaked from underground storage tanks and entered the groundwater, you may drink water from a well contaminated with TPH. You may breathe in some of the TPH compounds evaporating from a spill or leak if you are in the area where an accidental release has occurred. Children may be exposed by playing in soil contaminated with TPH. For more information on how you may be exposed to TPH, see Chapter 5.

1.4 HOW CAN TPH ENTER AND LEAVE MY BODY?

TPH can enter and leave your body when you breathe it in air; swallow it in water, food, or soil; or touch it. Most components of TPH will enter your bloodstream rapidly when you breathe them as a vapor or mist or when you swallow them. Some TPH compounds are widely distributed by the blood throughout your body and quickly break down into less harmful chemicals. Others may break down into more harmful chemicals. Other TPH compounds are slowly distributed by the blood to other parts of the body and do not readily break down. When you touch TPH compounds, they are absorbed more slowly and to a lesser extent than when you breathe or swallow them. Most TPH compounds leave your body through urine or when you exhale air containing the compounds. For more information on how TPH can enter and leave your body, see Chapter 6.

1.5 HOW CAN TPH AFFECT MY BODY?

Health effects from exposure to TPH depend on many factors. These include the types of chemical compounds in the TPH, how long the exposure lasts, and the amount of the chemicals contacted. Very little is known about the toxicity of many TPH compounds. Until more information is available, information about health effects of TPH must be based on specific compounds or petroleum products that have been studied.

The compounds in different TPH fractions affect the body in different ways. Some of the TPH compounds, particularly the smaller compounds such as benzene, toluene, and xylene (which are present in gasoline), can affect the human central nervous system. If exposures are high enough, death can occur. Breathing toluene at concentrations greater than 100 parts per million (100 ppm) for more than several hours can cause fatigue, headache, nausea, and drowsiness. When exposure is stopped, the symptoms will go away. However, if someone is exposed for a long time, permanent damage to the central nervous system can occur. One TPH compound (*n*-hexane) can affect the central nervous system in a different way, causing a nerve disorder called “peripheral neuropathy” characterized by numbness in the feet and legs and, in severe cases, paralysis. This has occurred in workers exposed to 500-2,500 ppm of *n*-hexane in the air. Swallowing some petroleum products such as gasoline and kerosene causes irritation of the throat and stomach, central nervous system depression, difficulty breathing, and pneumonia from breathing liquid into the lungs. The compounds in some TPH fractions can also affect the blood, immune system, liver, spleen, kidneys, developing fetus, and lungs. Certain TPH compounds can be irritating to the skin and eyes. Other TPH compounds, such as some mineral oils, are not very toxic and are used in foods.

To protect the public from the harmful effects of toxic chemicals and to find ways to-treat people who have been harmed, scientists use many tests.

One way to see if a chemical will hurt people is to learn how the chemical is absorbed, used, and released by the body; for some chemicals, animal testing may be necessary. Animal testing may

also be used to identify health effects such as cancer or birth defects. Without laboratory animals, scientists would lose a basic method to get information needed to make wise decisions to protect public health. Scientists have the responsibility to treat research animals with care and compassion. Laws today protect the welfare of research animals, and scientists must comply with strict animal care guidelines. Animal studies have shown effects on the lungs, central nervous system, liver, kidney, developing fetus, and reproductive system from exposure to TPH compounds, generally after breathing or swallowing the compounds.

One TPH compound (benzene) has been shown to cause cancer (leukemia) in people. The International Agency for Research on Cancer (IARC) has determined that benzene is carcinogenic to humans (Group 1 classification). Some other TPH compounds or petroleum products, such as benzo(a)pyrene and gasoline, are considered to be probably and possibly carcinogenic to humans (IARC Groups 2A and 2B, respectively) based on cancer studies in people and animals. Most of the other TPH compounds and products are considered not classifiable (Group 3) by IARC. See Chapter 6 for more information on how TPH can affect your body.

1.6 IS THERE A MEDICAL TEST TO DETERMINE IF I HAVE BEEN EXPOSED TO TPH?

There is no medical test that shows if you have been exposed to TPH. However, there are methods to determine if you have been exposed to some TPH compounds, fractions, or petroleum products. For example, a breakdown product of *n*-hexane can be measured in the urine. Benzene can be measured in exhaled air and a metabolite of benzene, phenol, can be measured in urine to show exposure to gasoline or to the TPH fraction containing benzene. Exposure to kerosene or gasoline can be determined by its smell on the breath or clothing. Methods also exist to determine if you have been exposed to other TPH compounds. For example, ethylbenzene can-be measured in the blood, urine, breath, and some body tissues of exposed people. However, many of these tests may not be available in your doctor’s office.

If you have TPH compounds in your body, they could be from exposure to many different products, and tests cannot determine exactly what you were exposed to. Tests are useful if you

suspect that you were exposed to a particular product or waste that contains TPH. More information on testing for TPH can be found in Chapter 3. For information on tests for exposure to specific TPH compounds, see the ATSDR toxicological profiles for benzene, toluene, total xylenes, polycyclic aromatic hydrocarbons, and hexane.

1.7 WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?

The federal government develops regulations and guidelines to protect public health. Regulations can be enforced by law. Federal agencies that develop regulations for toxic substances include the EPA, the NRC (Nuclear Regulatory Commission), the Occupational Safety and Health Administration (OSHA), and the Food and Drug Administration (FDA). Recommendations provide valuable guidelines to protect public health but cannot be enforced by law. Federal organizations that develop recommendations for toxic substances include the Agency for Toxic Substances and Disease Registry (ATSDR), Centers for Disease Control and Prevention (CDC), and the National Institute for Occupational Safety and Health (NIOSH).

Regulations and recommendations can be expressed in not-to-exceed levels in air, water, soil, or food that are usually based on levels that affect animals. Then they are adjusted to help protect people. Sometimes these not-to-exceed levels differ among federal organizations because of different exposure times (an 8-hour workday or a 24-hour day), the use of different animal studies, or other factors.

Recommendations and regulations are also periodically updated as more information becomes available. For the most current information, check with the federal agency or organization that provides it.

Although there are no federal regulations or guidelines for TPH in general, the government has developed regulations and guidelines for some of the TPH fractions and compounds. These are designed to protect the public from the possible harmful health effects of these chemicals. To

protect workers, the Occupational Safety and Health Administration (OSHA) has set a legal limit of 500 parts of petroleum distillates per million parts of air (500 ppm) in the workplace.

EPA regulates certain TPH fractions, products, or wastes containing TPH, as well as some individual TPH compounds. For example, there are regulations for TPH as oil; these regulations address oil pollution prevention and spill response, stormwater discharge, and underground injection control. EPA lists certain wastes containing TPH as hazardous. EPA also requires that the National Response Center be notified following a discharge or spill into the environment of 10 pounds or more of hazardous wastes containing benzene, a component in some TPH mixtures.

Nearly all states have cleanup standards for TPH or components of TPH (common cleanup standards are for gasoline, diesel fuel, and waste oil). Analytical methods are specified, many of which are considered to be TPH methods.

1.8 WHERE CAN I GET MORE INFORMATION?

If you have any more questions or concerns, please contact your community or state health or environmental quality department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road NE, Mailstop E-29
Atlanta, GA 30333

* Information line and technical assistance

Phone: l-888-42-ATSDR (l-888-422-8737)
Fax: (404) 639-6314 or 6324

ATSDR can also tell you the location of occupational and environmental health clinics. These clinics specialize in recognizing, evaluating, and treating illnesses resulting from exposure to hazardous substances.

* To order toxicological profiles, contact:

National Technical Information Service
5285 Port Royal Road
Springfield, VA 22 16 1
Phone: (800) 553-6847 or (703) 487-4650

2. OVERVIEW OF TOTAL PETROLEUM HYDROCARBONS

This document presents information in a way that is more summary in nature than the usual comprehensive toxicological profile. Total petroleum hydrocarbons (TPH) is such a broad family of compounds that it would be a large undertaking to present comprehensive environmental, chemical/physical, and health information on all the individual chemical components or on all petroleum products. This and subsequent chapters are designed to aid the reader in understanding what TPH is, what we know about it, the chance of significant exposure, and possible health consequences. Appendices are provided that present more detailed information.

2.1 DEFINITION OF TOTAL PETROLEUM HYDROCARBONS

TPH is defined as the measurable amount of petroleum-based hydrocarbon in an environmental media. It is, thus, dependent on analysis of the medium in which it is found (Gustafson 1997). Since it is a measured, gross quantity without identification of its constituents, the TPH "value" still represents a mixture. Thus, TPH itself is not a direct indicator of risk to humans or to the environment. The TPH value can be a result from one of several analytical methods, some of which have been used for decades and others developed in the past several years. Analytical methods are evolving in response to needs of the risk assessors. In keeping with these developments, definition of TPH by ATSDR is closely tied to analytical methods and their results. The ATSDR approach to assessing the public health implications of exposure to TPH is presented in Section 2.3.

There are several hundred individual hydrocarbon chemicals defined as petroleum-based, with more than 2.50 petroleum components identified in Appendix D of this profile. Further, each petroleum product has its own mix of constituents. One reason for this is that crude oil, itself, varies in its composition. Some of this variation is reflected in the finished petroleum product. The acronym PHC (petroleum hydrocarbons) is widely used to refer to the hydrogen- and carbon-containing compounds originating from crude oil, but PHC should be distinguished from TPH, because TPH is specifically associated with environmental sampling and analytical results.

Petroleum crude oils can be broadly divided into paraffinic, asphaltic, and mixed crude oils (WHO 1982). Paraffinic crude oils are composed of aliphatic hydrocarbons (paraffins), paraffin wax (longer chain

aliphatics), and high grade oils. Naphtha is the lightest of the paraffin fraction, followed by kerosene fractions. Asphaltic crude oils contain larger concentrations of cycloaliphatics and high viscosity lubricating oils. Petroleum solvents are the product of crude oil distillation and are generally classified by boiling point ranges. Lubricants, greases, and waxes are high boiling point fractions of crude oils. The heaviest, solid fractions of crude oils are the residuals or bitumen.

Some products are highly predictable (e.g., jet fuels) with specific fractions of defined components; others, for example, automotive gasolines, contain broader ranges of hydrocarbon types and amounts. Table D- 1 in Appendix D provides a comprehensive list of petroleum hydrocarbons.

Petroleum products, themselves, are the source of the many components, but do not define what is TPH. They help define the potential hydrocarbons that become environmental contaminants, but any ultimate exposure is determined also by how the product changes with use, by the nature of the release, and by the hydrocarbon's environmental fate. When petroleum products are released into the environment, changes occur that significantly affect their potential effects. Physical, chemical, and biological processes change the location and concentration of hydrocarbons at any particular site.

Petroleum hydrocarbons are commonly found environmental contaminants, though they are not usually classified as hazardous wastes. Many petroleum products are used in modern society, including those that are fundamental to our lives (i:e., transportation fuels, heating and power-generating fuels). The volume of crude oil or petroleum products that is used today dwarfs all other chemicals of environmental and health concern. Due to the numbers of facilities, individuals, and processes and the various ways the products are stored and handled, environmental contamination is potentially widespread.

Soil and groundwater petroleum hydrocarbon contamination has long been of concern and has spurred various analytical and site remediation developments, e.g., risk-based corrective actions (ASTM's Risk-Based Corrective Action [RBCA]), EPA and state government underground storage tank (UST) programs, British Columbia's Ministry of Environment's development of remediation criteria for petroleum contamination (primarily environmental risks) (BC 1995), and the annual Amherst Massachusetts conference from which the Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG) was formed. The TPHCWG is made up of industry, government, and academic scientists, working to develop a broad set of guidelines to be used by engineering and public health

professionals in decisions on petroleum contaminated media. In 1997 the criteria working group published a technical overview of their risk management approach to TPH (TPHCWG 1997a), which represents the most comprehensive effort in this area to date. In 1997 the TPHCWG published two volumes, *Selection of Representative TPH Fractions Based on Fate and Transport Considerations* (Vol. 3) and *Development of Fraction Specific Reference Doses (RfDs) and Reference Concentrations (RfCs) for Total Petroleum Hydrocarbons (TPH)* (Vo1.4) (TPHCWG 1997b, 1997c). In 1998 the TPHCWG published Volume 1*, Analysis of Petroleum Hydrocarbons in Environmental Media* (TPHCWG 1998a) and Volume 2*, Composition of Petroleum Mixtures* (TPHCWG 1998b).

2.2 TOTAL PETROLEUM HYDROCARBONS ANALYSIS OVERVIEW

The TPH method of analysis often used, and required by many regulatory agencies, is EPA Method 4 18.1. This method provides a "one number" value of TPH in an environmental media; it does not provide information on the composition (i.e., individual constituents of the hydrocarbon mixture). The amount of TPH measured by this method depends on the ability of the solvent used to extract the hydrocarbon from the environmental media and the absorption of infrared (IR) light by the hydrocarbons in the solvent extract. EPA Method 418.1 is not specific to hydrocarbons and does not always indicate petroleum contamination (e.g., humic acid, a non-petroleum hydrocarbon, may be detected by this method).

An important feature of the TPH analytical methods is the use of an Equivalent Carbon Number Index (EC). The EC represents equivalent boiling points for hydrocarbons and is the physical characteristic that is the basis for separating petroleum (and other) components in chemical analysis. Petroleum fractions as discussed in this profile are defined by EC.

Another analytical method commonly used for TPH is EPA Method 8015 Modified. This method reports the concentration of purgeable and extractable hydrocarbons; these are sometimes referred to as gasoline and diesel range organics, GRO and DRO, respectively, because the boiling point ranges of the hydrocarbon in each roughly correspond to those of gasoline (C_6 to C_{10-12}) and diesel fuel (C_{8-12} to C_{24-26}), respectively. Purgeable hydrocarbons are measured by purge-and-trap gas chromatography (GC) analysis using a flame ionization detector (FID), while the extractable hydrocarbons are extracted and concentrated prior to analysis by GUFID. The results are most frequently reported as

single numbers for purgeable and extractable hydrocarbons. Before the TPHCWG began publishing its TPH guides, the Massachusetts Department of Environmental Protection (MADEP) developed risk assessment and analytical methodologies for TPH (Hutcheson et al. 1996). MADEP developed a method based on EPA Method 801.5 Modified which gives a measure of the aromatic and aliphatic content of the hydrocarbon in each of several carbon number ranges (fractions). The MADEP method is based on standard EPA methods, which allows it to be easily implemented by laboratories, though there are limitations with the method (see Section 3.3). EPA has proposed a modification in its test procedure for analysis of "oil and grease and total petroleum hydrocarbons" that not only overcomes the problem of using freon as a solvent, but also provides more refined separation of aliphatic and aromatic fractions (EPA 1998a).

The Risk-Based Corrective Action (RBCA) guidance of American Society for Testing and Materials (ASTM), published in 1995, is an important document for public and private institutions that remediate petroleum contaminated sites (ASTM 1995). EPA is telling agencies implementing risk- based decision-making that the ASTM standard may be a good starting point for risk management (EPA 1995c).

2.3 TPH FRACTIONS AND THE ATSDR APPROACH TO EVALUATING THE PUBLIC HEALTH IMPLICATIONS OF EXPOSURE TO TPH

The public health implications associated with TPH are common to the broader questions of chemical mixtures. What does one know about the makeup and adverse health effects associated with the whole mixture? Does one select the most toxic or carcinogenic elements or representative chemical(s), or does one rely on whole product toxicity results? In the case of TPH, one sample is likely to vary significantly in content from other samples, even with similar "single value" results.

This profile builds on the efforts by the TPHCWG and MADEP to group chemicals into fractions with similar environmental transport characteristics (i.e., transport fractions). An important difference is ATSDR's concern with all possible exposure periods, from acute through chronic, whereas other agencies or groups have focused on longer-term exposures. The common characteristic of all of these approaches is the attempt to gather the available information about the toxicity and the risks associated with transport fractions.

Although chemicals grouped by transport fraction generally have similar toxicological properties, this is not always the case. For example, benzene is a carcinogen, but toluene, ethylbenzene, and xylenes are not. However, it is more appropriate to group benzene with compounds that have similar environmental transport properties than to group it with other carcinogens such as benzo(a)pyrene that have very different environmental transport properties. Section 6.1.1 provides a more detailed discussion of the various transport fractions.

ATSDR's mission of providing public health support to communities with potential exposure to hazardous wastes is different from that of the ASTM, for example, which developed the RBCA guide for the purpose of remediation of petroleum-contaminated sites. Also, ecological risk assessment is a fundamental feature of the ASTM and British Columbia methodologies, though not for ATSDR.

Because a critical aspect of assessing the toxic effects of TPH is the measurement of the compounds, one must first appreciate the origin of the various fractions (compounds) of TPH. Transport fractions are determined by several chemical and physical properties (i.e. solubility, vapor pressure, and propensity to bind with soil and organic particles). These properties are the basis of measures of leachability and volatility of individual hydrocarbons and transport fractions. The TPHCWG approach defines petroleum hydrocarbon transport fractions by equivalent carbon number grouped into 13 fractions (see Section 6.1.2). The "analytical fractions" are then set to match these transport fractions, using specific *n*-alkanes to mark the analytical results for aliphatics and selected aromatics to delineate hydrocarbons containing benzene rings. ATSDR has used the basic TPHCWG approach and modified the fractional groups (see Chapter 6). Fate and transport considerations are discussed in more detail in Chapter 5. The TPHCWG transport fractions' physical properties are presented in Table 2-l.

The approach to evaluating the potential health effects for these transport fractions taken by ATSDR and the TPHCWG, however, uses a reduced number of fractions, namely three aliphatic fractions and three aromatic fractions. Health effects screening values based on representative chemicals or-mixtures for each of the fractions were developed using ATSDR minimal risk levels (MRLs). Table 2-2 presents the ATSDR TPH fractions and their representative compounds or mixtures. In general, the most toxic representative compound or mixture for each fraction is used to indicate the potential toxicity of the entire fraction. Selection of the representative compounds and mixtures is discussed in detail in Sections 6.2,

Table 2-1. Representative Physical Parameters for TPH Analytical Fractions Based on Correlation to Relative Boiling Point Index

Fraction	Solubility, mg/L	Vapor pressure, atm	Henry's law constant, cm^3/cm^3	Log K_{oc}
Aromatics				
EC_5–EC_7[a]	220	0.11	1.5	3
$EC_{>7}$–EC_8[b]	130	0.035	0.86	3.1
$EC_{>8}$–EC_{10}	65	0.0063	0.39	3.2
$EC_{>10}$–EC_{12}	25	0.00063	0.13	3.4
$EC_{>12}$–EC_{16}	5.8	0.000048	0.028	3.7
$EC_{>16}$–EC_{21}	0.65	0.0000011	0.0025	4.2
$EC_{>21}$–EC_{35}	0.0066	0.00000000044	0.000017	5.1
Aliphatics				
EC_5–EC_6	36	0.35	47	2.9
$EC_{>6}$–EC_8	5.4	0.063	50	3.6
$EC_{>8}$–EC_{10}	0.43	0.0063	55	4.5
$EC_{>10}$–EC_{12}	0.034	0.00063	60	5.4
$EC_{>12}$–EC_{16}	0.00076	0.000076	69	6.7
$EC_{>16}$–EC_{35}	0.0000025	0.0000011	85	8.8

[a] The only compound contained in this fraction is benzene.
[b] The only compound contained in this fraction is toluene.

EC = equivalent carbon number

Source: TPHCWG 1997b

Table 2-2. ATSDR TPH Fractions and Representative Compounds

Chemical fraction, in EC[a]	Representative compounds
Aromatics	
EC_6 - EC_9	Benzene, toluene, ethylbenzene, xylenes
$EC_{>9}$ - EC_{16}	Isopropyl benzene, naphthalene
$EC_{>16}$ - EC_{35}	Fluorene, fluoranthene, benzo(a)pyrene
Aliphatics	
EC_5 - EC_8	*n*-Hexane
$EC_{>8}$ - EC_{16}	JP-5, JP-7, JP-8, kerosene, dearomatized petroleum stream
$EC_{>16}$ - EC_{35}	Mineral oils

[a] EC = equivalent carbon number index. EC is based on equivalent retention times on a boiling point GC column, normalized to n-alkanes

6.3, and 6.6. In addition, existing cancer assessments for each fraction are presented and discussed in Chapter 6 and Appendix A.

Despite the large number of hydrocarbons found in petroleum products and the widespread nature of petroleum use and contamination, only a relatively small number of the compounds are well characterized for toxicity. The health effects of some fractions can be well characterized, based on their components or representative compounds (e.g., light aromatic fraction-BTEX-benzene, toluene, ethylbenzene, and xylenes). However, heavier TPH fractions have far fewer well characterized compounds. Systemic and carcinogenic effects are known to be associated with petroleum hydrocarbons, but ATSDR does not develop health guidance values for carcinogenic end points (ATSDR 1996b). See Chapter 6 for further discussion of the ATSDR approaches and the approaches of other groups (MADEP, TPHCWG, and ASTM).

3. IDENTITY AND ANALYSIS OF TOTAL PETROLEUM HYDROCARBONS

3.1 INTRODUCTION

Petroleum hydrocarbons (PHCs) are common site contaminants, but they are not generally regulated as hazardous wastes. Methods for sampling and analysis of environmental media for the family of PHCs are generally thought of as TPH methods. For purposes of this profile, the term TPH refers not only to analytical results, but also to environmental and health properties of PHCs. In part due to the complexity of TPH components themselves, little is known about their potential for health or environmental impacts. As gross measures of petroleum contamination, TPH results simply show that petroleum hydrocarbons are present in the sampled media. Measured TPH values suggest the relative potential for human exposure and, therefore, the relative potential for human health effects. The assessment of health effects due to TPH exposure requires much more detailed information than what is provided by a single TPH value. This chapter, Chapter 5, and the accompanying Appendix E provide more detailed physical and chemical properties and analytical information on TPH and its components.

The federal government has left much of the specific regulation and oversight of crude oil production/ refining to the states. Leaking underground storage tanks (LUST) are the most frequent causes of federal and state governmental involvement in petroleum hydrocarbon problems. Soil contamination has been a growing concern, because it can be a source of groundwater (drinking water) contamination; contaminated soils can reduce the usability of land for development; and weathered petroleum residuals may stay bound to soils for years. Positive TPH test results may require action on the part of land owners, local or state governments, and engineering firms called on to remove or reduce the TPH problem.

ATSDR has the responsibility for health assessment at National Priorities List (NPL) hazardous waste sites, many of which have petroleum hydrocarbon contamination. Specific contaminants that are components of TPH, such as BTEX (benzene, toluene, ethylbenzene, and xylene), *n*-hexane, jet fuels, fuel oils, and mineral-based crankcase oil, have been studied by ATSDR and a number of toxicological profiles have been developed on individual constituents and petroleum products. The

ATSDR profiles relevant to petroleum products are listed in Table 3- 1. However, TPH itself has not been as extensively studied by ATSDR and no previous profile was developed. Although several toxicological profiles have been developed for petroleum products and for specific chemicals found in petroleum, TPH test results have been too nonspecific to be of real value in the assessment of its potential health effects.

Several approaches are discussed in this document for interpreting TPH and related analytical results. The TPH approach taken by EPA and others, through the mid-1990s, followed general risk assessment approaches for chemical mixtures. In all approaches there is a need to reduce a comprehensive list of potential petroleum hydrocarbons to a manageable size. Depending on how conservative the approach is, methods that have been used select: (1) the most toxic among the TPH compounds (indicator approach); (2) one or more representative compounds (surrogate approach, but independent of relative mix of compounds); or (3) representative compounds for fractions of similar petroleum hydrocarbons. ATSDR has taken, in part, the third approach in keeping with the Total Petroleum Hydrocarbons Criteria Working Group (TPHCWG), but has developed its own set of TPH fraction representatives, many of which overlap those of the TPHCWG. In addition, this profile provides information on petroleum products, where such information exists. TPH risk (screening) values for fractions presented in this profile are based on the ATSDR MRLs previously developed for individual constituents and petroleum products. These MRLs are summarized in Appendix A. This fraction approach is the most demanding in information gathering and because of that would appear to be the most rigorous approach to date. Sections 6.1.2 and 6.1.3 contain a more comprehensive discussion of the approaches. The identity, chemical-physical, and analytical information discussed and listed in this chapter, in Appendices D and E, and in Chapter 5 are integral to defining TPH.

3.2 CHEMICAL AND PHYSICAL INFORMATION

Petroleum products are complex mixtures of hundreds of hydrocarbon compounds, ranging from light, volatile, short-chained organic compounds to heavy, long-chained, branched compounds. The exact composition of petroleum products varies depending upon (1) the source of the crude oil (crude oil is derived from underground reservoirs which vary greatly in their chemical composition) and (2) the refining practices used to produce the product.

Table 3-1. ATSDR Profiles with Information on Analytical Methods Relevant to Petroleum Products

Petroleum product	Reference
Jet Fuels, JP-4/JP-7	ATSDR 1995c
Jet Fuels, JP-5/JP-8	ATSDR 1998b
Stoddard solvent	ATSDR 1995b
Automotive gasoline	ATSDR 1995a
Fuel oils	ATSDR 1995g
Crankcase oil	ATSDR 1997c
Benzene	ATSDR 1997a
Toluene	ATSDR 1994
Ethylbenzene	ATSDR 1999a
Xylenes	ATSDR 1995d
Naphthalene	ATSDR 1995e
Methyl *t*-butyl ether	ATSDR 1996a
Polycyclic aromatic hydrocarbons	ATSDR 1995f
Hexane	ATSDR 1999b
Mineral oil (hydraulic fluids)	ATSDR 1997b

3. IDENTITY AND ANALYSIS OF TOTAL PETROLEUM HYDROCARBONS

During the refining process, crude oil is separated into fractions having similar boiling points. These fractions are then modified by cracking, condensation, polymerization, and alkylation processes, and are formulated into commercial products such as naphtha, gasoline, jet fuel, and fuel oils. The composition of any one of these products can vary based on the refinery involved, time of year, variation in additives or modifiers, and other factors. The chemical composition of the product can be further affected by weathering and/or biological modification upon release to the environment. The following subsections present overviews of petroleum products. Also, a master list of individual aliphatic and aromatic compounds found in TPH is provided in Appendix D. Further information on whole petroleum products, their identity, major components, and physical/chemical properties is found in Appendix E.

Automotive Gasoline. Automotive gasoline is a mixture of low-boiling hydrocarbon compounds suitable for use in spark-ignited internal combustion engines and having an octane rating of at least 60. Additives that have been used in gasoline include alkyl tertiary butyl ethers (e.g. MTBE), ethanol (ethyl alcohol), methanol (methyl alcohol), tetramethyl-lead, tetraethyl-lead, ethylene dichloride, and ethylene dibromide.

Other categories of compounds that may be added to gasoline include anti-knock agents, antioxidants, metal deactivators, lead scavengers, anti-rust agents, anti-icing agents, upper-cylinder lubricants, detergents, and dyes (ATSDR 1995a).

Automotive gasoline typically contains about 150 hydrocarbon compounds, though nearly 1,000 have been identified (ATSDR 1995a). The relative concentrations of the compounds vary considerably depending on the source of crude oil, refinery process, and product specifications. Typical hydrocarbon chain lengths range from C_4 through C_{l2} with a general hydrocarbon distribution consisting of 4-8% alkanes, 2-5% alkenes, 25-40% isoalkanes, 3-7% cycloalkanes, l-4% cycloalkenes, and 20-50% aromatics (IARC 1989a). However, these proportions vary greatly. Unleaded gasolines may have higher proportions of aromatic hydrocarbons than leaded gasolines.

Table E-1.b (Appendix E) presents ranges and weight percentage means for a representative subset of the hydrocarbon compounds identified in gasoline. In cases where data are not available, the range and mean are left blank.

Stoddard Solvent. Stoddard solvent is a petroleum distillate widely used as a dry cleaning solvent and as a general cleaner and degreaser. It may also be used as a paint thinner, as a solvent in some types of photocopier toners, in some types of printing inks, and in some adhesives. Stoddard solvent is considered to be a form of mineral spirits, white spirits, and naphtha; however, not all forms of mineral spirits, white spirits, and naphtha are considered to be Stoddard solvent (ATSDR 1995b).

Stoddard solvent consists of 30-50% linear and branched alkanes, 30-40% cycloalkanes, and lo-20% aromatic hydrocarbons. Its typical hydrocarbon chain ranges from C_7 through C_{12} in length.

Although a complete list of the individual compounds comprising Stoddard solvent is not available (Air Force 1989) some of the major components are presented in Table E-2.b (Appendix E). Alcohols, glycols, and ketones are not included in the composition, as few, if any, of these types of compounds would be expected to be present in Stoddard solvent (ATSDR 1995b). Possible contaminants may include lead (<1 ppm) and sulfur (3.5 ppm).

Jet Fuel. Jet fuels are light petroleum distillates that are available in several forms suitable for use in various types ofjet engines. The exact compositions of jet fuels are established by the U.S. Air Force, using specifications that yield maximum performance by the aircraft. The major jet fuels used by the military are JP-4, JP-5, JP-6, JP-7, and JP-8. Briefly, JP-4 is a wide-cut fuel developed for broad availability in times of need. JP-6 is a higher cut than JP-4 and is characterized by fewer impurities. JP-5 is specially blended kerosene, and JP-7 is a high flash point special kerosene used in advanced supersonic aircraft. JP-8 is a kerosene modeled on Jet A-l fuel (used in civilian aircraft). For this profile, JP-4 will be used as the prototype jet fuel due to its broad availability and extensive use.

Typical hydrocarbon chain lengths characterizing JP-4 range from C_4 to C_{16}. Aviation fuels consist primarily of straight and branched alkanes and cycloalkanes. Aromatic hydrocarbons are limited to 20-25% of the total mixture because they produce smoke when burned. A maximum of5% alkenes are allowed in JP-4 (ATSDR 1995c). The approximate distribution by chemical class is: 32% straight alkanes, 31% branched alkanes, 16% cycloalkanes, and 21% aromatic hydrocarbons (ABB Environmental 1990). The typical hydrocarbon composition of JP-4 is presented in Table E-3.b (Appendix E).

Fuel Oil #1. Fuel oil #l is a petroleum distillate that is one of the most widely used of the fuel oil types. It is used in atomizing burners that spray fuel into a combustion chamber where the tiny droplets bum while in suspension. It is also used as a carrier for pesticides, as a weed killer, as a mold release agent in the ceramic and pottery industry, and in the cleaning industry. It is found in asphalt coatings, enamels, paints, thinners, and varnishes.

Fuel oil #1 is a light petroleum distillate (straight-run kerosene) consisting primarily of hydrocarbons in the range C_9-C_{16} (ATSDR 19958). Fuel oil #l is very similar in composition to diesel fuel oil #l; the primary difference is in the additives. The typical hydrocarbon composition of fuel oil #l is presented in Table E-4.b (Appendix E).

Fuel Oil #2. Fuel oil #2 is a petroleum distillate that may be referred to as domestic or industrial. The domestic fuel oil #2 is usually lighter and straight-run refined; it is used primarily for home heating and to produce diesel fuel #2. Industrial distillate is the cracked type, or a blend of both. It is used in smelting furnaces, ceramic kilns, and packaged boilers (ABB Environmental 1990).

Fuel oil #2 is characterized by hydrocarbon chain lengths in the C_{11}-C_{20} range, whereas diesel fuels predominantly contain a mixture of C_{10}-C_{19} hydrocarbons (ATSDR 1995g). The composition consists of approximately 64% aliphatic hydrocarbons (straight chain alkanes and cycloalkanes), l-2% unsaturated hydrocarbons (alkenes), and 35% aromatic hydrocarbons (including alkylbenzenes and 2-, 3-ring aromatics) (Air Force 1989). Fuel oil #2 contains less than 5% polycyclic aromatic hydrocarbons (IARC 1989b). The typical hydrocarbon composition of fuel oil #2 is presented in Table E-4.b (Appendix E).

Fuel Oil #6. Fuel oil #6 is also called Bunker C or residual. It is the residual from crude oil after the light oils, gasoline, naphtha, fuel oil #l, and fuel oil #2 have been fractioned off. Fuel oil #6 can be blended directly to heavy fuel oil or made into asphalt. It is limited to commercial and industrial uses where sufficient heat is available to fluidize the oil for pumping and combustion (ABB Environmental 1990).

Residual fuel oils are generally more complex in composition and impurities than distillate fuels. Limited data are available on the composition of fuel oil #6 (ATSDR 1995g). Clark et al. (1990) indicate that fuel oil #6 includes about 25% aromatics, 15% paraffins, 45% naphthenes, and 15% non-hydrocarbon

compounds. Polycyclic aromatic hydrocarbons (PAHs) and alkyl PAHs and metals are important hazardous and persistent components of fuel oil #6. Table E-4.b (Appendix E) presents the results of an analysis of one sample (Pancirov and Brown 1975).

Mineral Oils, Including Mineral-based Crankcase Oil. Mineral oils are often lubricating oils, but they also have medicinal and food uses. A major type of hydraulic fluid is the mineral oil class of hydraulic fluids (ATSDR 1997b). The mineral-based oils are produced from heavy-end crude oil distillates. Distillate streams may be treated in several ways, such as vacuum-, solvent-, acid-, or hydro- treated, to produce oils with commercial properties. Hydrocarbon numbers ranging from C_{15} to C_{50} are found in the various types of mineral oils, with the heavier distillates having higher percentages of the higher carbon number compounds (IARC 1984).

Crankcase oil or motor oil may be either mineral-based or synthetic. The mineral-based oils are more widely used than the synthetic oils and may be used in automotive engines, railroad and truck diesel engines, marine equipment, jet and other aircraft engines, and most small 2- and 4-stroke engines.

The mineral-based oils contain hundreds to thousands of hydrocarbon compounds, including a substantial fraction of nitrogen- and sulfur-containing compounds. The hydrocarbons are mainly mixtures of straight and branched chain hydrocarbons (alkanes), cycloalkanes, and aromatic hydrocarbons. PAHs, alkyl PAHs, and metals are important components of motor oils and crankcase oils, with the used oils typically having higher concentrations than the new unused oils. Typical carbon number chain lengths range from C_{l5} to C_{50} (ABB Environmental 1990).

Because of the wide range of uses and the potential for close contact with the engine to alter oil composition, the exact composition of crankcase oil/motor oil has not been specifically defined. Table E-5.b (Appendix E) presents analytical results for some constituents in used automotive oil (ABB Environmental 1990).

3.3 ANALYTICAL METHODS

The purpose of this section is to describe well established analytical methods that are available for detecting, and/or measuring, and/or monitoring TPH and its metabolites, as well as other biomarkers of exposure and effect of TPH. The intent is not to provide an exhaustive list of analytical methods. Rather, the intention is to identify well-established methods that are used as the standard methods approved by federal agencies and organizations such as EPA and the National Institute for Occupational Safety and Health (NIOSH) or methods prescribed by state governments for water and soil analysis. Other methods presented are those that are approved by groups such as ASTM.

The term “total petroleum hydrocarbons” (TPH) is generally used to describe the measurable amount of petroleum-based hydrocarbons in the environment; and thus the TPH information obtained depends on the analytical method used. One of the difficulties with TPH analysis is that the scope of the methods varies greatly. Some methods are nonspecific while others provide results for hydrocarbons in a boiling point range. Interpretation of analytical results requires an understanding of how the determination was made.

Analytical methods for some petroleum products are discussed in existing ATSDR toxicological profiles. The very volatile gases (compounds with 4 carbons or less), crude oil, and the solid bituminous materials such as asphalt are not included in this discussion of analytical methods. ATSDR profiles relevant to petroleum products are listed in Table 3-1. The TPHCWG also addresses some of these issues from a different perspective which includes, in some cases, more detail and references than provided here (TPHCWG 1998a).

3.3.1 Environmental Samples.

Most of the analytical methods discussed here for TPH have been developed within the framework of federal and state regulatory initiatives. The initial implementation of the Federal Water Pollution Control Act (FWPCA) focused on controlling conventional pollutants such as oil and grease. Methods developed for monitoring wastewaters included EPA Method 4 13.1 (EPA 1979a) and EPA Method 413.2 (EPA 1979d) for Total Recoverable Oil and Grease (TOG), and EPA Method 418.1 for Total Recoverable Petroleum Hydrocarbons (TRPH) (EPA 1979c). Freon-extractable material is reported as TOG. Polar components may be removed by treatment with silica gel, and the material remaining,

as determined by infrared (IR) spectrometry, is defined as Total Recoverable Petroleum Hydrocarbons (TPH, TRPH, or TPH-IR). A number of modifications of these methods exist. EPA Method 418.1 has been one of the most widely used methods for the determination of TPH in soils. Many states use, or permit the use of, EPA Method 418.1 for identification of petroleum products and during remediation of sites (George 1992; Judge et al. 1997, 1998). This method is subject to limitations, such as inter-laboratory variations and inherent inaccuracies (George 1992). In addition, the EPA proposed to withdraw wastewater methods which use Freon- 113 extraction (EPA 1996a). These methods will be replaced with EPA Method 1664: n-Hexane Extractable Material (HEM) and Silica Gel Treated *n*-Hexane Extractable Material (SGT-HEM) by Extraction and Gravimetry (Oil and Grease and Total Petroleum Hydrocarbons) (EPA 1996a). Conventional methods of TPH analysis are summarized in Table 3-2.

These conventional TPH analytical methods have been used widely to investigate sites that may be contaminated with petroleum hydrocarbon products. Many state and local regulatory agencies rely on and require EPA Method 418.1 (EPA 1979c) for determination of petroleum hydrocarbons (Murray 1994). The important advantages of this approach are (1) the method is relatively inexpensive, and (2) excellent sample reproducibility can be obtained. The disadvantages are (1) petroleum hydrocarbon composition varies among sources and over time, so results are not always comparable; (2) the more volatile compounds in gasoline and light fuel oil may be lost in the solvent concentration step; (3) there are inherent inaccuracies in the method; and (4) the method provides virtually no information on the types of hydrocarbons present. Several recent reports have detailed the problems with this approach (George 1992; Rhodes et al. 1995/1996). Thus, these conventional TPH methods, although they provide adequate screening information, do not provide sufficient information on the extent of the contamination and product type. In addition, The Clean Air Act Amendments of 1990 require the phaseout of the use of chlorofluorocarbons. Therefore, the EPA methods using Freon-l 13 will be replaced with EPA Method 1664, *n*-Hexane Extractable Material (HEM) and Silica Gel Treated *n*-Hexane Extractable Material (SGT-HEM) by Extraction and Gravimetry (EPA 1996a). Proposed Method 1664 includes thorough method quality control, but results may not equivalent to the current methods. Examples of TPH methods for environmental media are shown in Table 3-3.

Gas chromatography (GC) methods do provide some information about the product type. Most methods involve a sample preparation procedure followed by analysis using GC techniques. GC

Table 3-2. Summary of Conventional TPH Methods

Method name	Type	Matrix	Scope of method	Reference
Gravimetric methods				
EPA Method 413.1	TOG	Water and wastewater	Petroleum fuels from gasoline through #2 fuel oils are completely or partially lost in the solvent removal operation. Recoveries of some crude oils and heavy fuel oils will be low.	EPA 1979a
EPA Method 9070	TOG	Solid waste	Applicable to determination of relatively nonvolatile hydrocarbons. Not applicable to measurement of light hydrocarbons; petroleum fuels, from gasoline through No. 2 fuel oils, are completely or partially lost. Recoveries of some crude oils and heavy fuel oils will be low.	EPA 1995d
EPA Method 9071A	TOG	Sludge	Used to recover low levels of oil and grease from sludge. Used when relatively polar, heavy petroleum fractions are present, or when the levels of nonvolatile greases challenge the solubility limit of the solvent. Not recommended for measurement of low-boiling fractions.	EPA 1995d
Standard Method 5520B	TOG	Water and wastewater	Suitable for biological lipids and mineral hydrocarbons. Not suitable for low-boiling fractions.	APHA 1992
Standard Method 5520D	TOG	Water and wastewater	Suitable for biological lipids and mineral hydrocarbons. Method D is the method of choice when relatively polar, heavy petroleum fractions are present, or when the levels of nonvolatile greases may challenge the solubility of the solvent	APHA 1992
Standard Method 5520E	TOG	Sludges	Suitable for biological lipids and mineral hydrocarbons. Not suitable for low-boiling fractions. Method E is a modification of Method D.	APHA 1992
Standard Method 5520F	TPH	Water and wastewater	Suitable for biological lipids and mineral hydrocarbons. Not suitable for low-boiling fractions. May be used in conjunction with Method B, D or E.	APHA 1992
Infrared methods				
EPA Method 413.2	TOG	Water and wastewater	Applicable to the measurement of light fuels, although loss of about half of any gasoline present during the extraction manipulation can be expected	EPA 1979c

Table 3-2. Summary of Conventional TPH Methods (continued)

Method name	Type	Matrix	Scope of method	Reference
EPA Method 418.1	TPH	Water and wastewater	Applicable to a wide range of hydrocarbons, although volatile components will be lost. Modifications exist for measurement of TPH in soil.	EPA 1979c
Standard Method 5520C	TOG	Water and wastewater	Suitable for biological lipids and mineral hydrocarbons. The lighter distillates, with the exception of gasoline, may be measured accurately. Method C is the method of choice for low levels of oil and grease.	APHA 1992

TOG = (Total) oil and grease; TPH = Total Petroleum Hydrocarbons.

Table 3-3. Analytical Methods for Determining Total Petroleum Hydrocarbons in Environmental Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Occupational air (hydrocarbons, BP=36–126 °C)	Sample collection on solvent tubes; solvent desorption	GC/FID	0.001 to 0.01 mg per sample[a]	>75[b]	NIOSH 1994 (Method 1500)
Ambient air	None	Continuous sampling/FID	0.16 ppm (0.1 mg/m^3)	Not reported	Lodge 1988 (ISC Method 108)
Ambient air	Collection on Tenax GC cartridge; thermal desorption	Capillary GC/MS	~20 ng injected	Not reported	EPA 1988 (Method TO-1)
Water, wastes (Oil and grease)	Solvent extraction	Gravimetric			EPA 1979e (Method 413.1)
Water, wastes (Oil and grease)	Solvent extraction	IR	~0.2 mg/L	99	EPA 1979d (Method 413.2)
Water and wastes (TRPH)	Solvent extraction; silica gel column separation	IR	≤1 mg/L		EPA 1979c (Method 418.1)
Water, aqueous wastes (oil and grease)	Solvent extraction	Gravimetric	5 mg/L	93	EPA 1988a (Method 9070)
Sludge and sediment (oil and grease)	Sample is dried; Soxhlet extraction	Gravimetric	10 mg/L	93	EPA 1988a (Method 9071A)
Soils, sediments, fly ash (TRPH)	Supercritical fluid extraction	Method 8015B	See Method 8015B	See Method 8015B	EPA 1995d (Method 3560)
Soil (TPH)	Extraction; filtration	Immunoassay	5.8 ppm	Not reported[d]	EPA 1995d (Method 4030)
Ground or surface water, soil (DRO, GRO)	DRO: solvent extraction; GRO: purge and trap or vacuum distillation or headspace sampling	Capillary GC/FID	DRO (low aromatic) ≤75 ppm; (regular)≤25 ppm	DRO: (low aromatic) 72–96; (regular) 125	EPA 1995d (Method 8015B)
Water (petroleum oils)	Solvent extraction; evaporation	GC/FID	Not reported[c]	Not reported[c]	ASTM 1994 (Method D 3328)
	Solvent extraction; evaporation	IR	Not reported[c]	Not reported[c]	ASTM 1994 (Method D 3414)

Table 3-3. Analytical Methods for Determining Total Petroleum Hydrocarbons in Environmental Samples (continued)

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Soils	Supercritical fluid extraction	IR		82.9	Lopez-Avila et al. 1993
Soils (gasoline and diesel)	Solvent extraction	Capillary GC/MSD-SIM	≤1 ppm	80–120	Xiang et al. 1995
Fish tissue (kerosene range)	Homogenization; digestion/solvent extraction	Capillary GC/FID	Low to sub-ppm	95	Guiney et al. 1987
Fish tissue	Solvent extraction; fractionation on silica gel columns; solvent exchange	GC/FID	Low ppm	90–113	Murray and Lockhart 1981
Avian tissue (liver, kidney, fat, brain tissue	Solvent extraction; saponification of fatty tissue; clean-up on adsorbent columns	Capillary GC/FID and GC/MS	ppb range	40–100 (target compounds)	Gay et al. 1980
Marine lipids (fish oils)	Saponification of lipids, followed by solvent extraction; cleanup on adsorbent columns or plates	GC/FID	Not reported[a]	Not reported[a]	Farrington et al. 1973
Mollusc tissue (TPH)	Homogenization; digestion and solvent extraction; fractionation on an adsorbent column	Capillary GC/FID	≈0.1 ppm	Not reported	Steimle et al 1986
Fish tissue, fish liver	Solvent extraction, followed by saponification of lipids; fractionation on adsorbent columns	Capillary GC/FID; confirmation by capillary GC/MS	Low ppm	Not reported	Fowler et al. 1993
Palm kernel oil (hydrocarbons)	Kernels ground, pressed, filtered; Soxhlet extraction of oil; fractionation on silica gel columns	GC/FID; identification of components by GC/MSD	≈1 µg/g	70–87	Tan and Kuntom 1994

[a] Actual detection limits will depend upon the volume of air sampled
[b] Desorption efficiency
[c] Method is qualitative; samples are compared to known petroleum oils
[d] Method 4030 provides an estimated of concentration

BP = boiling point; DRO = diesel range organics; GC = gas chromatography; GRO = gasoline range organics; FID = flame ionization detector; IR = infrared spectrophotometry; MS = mass spectrometry; MSD = mass selective detector; SIM = selected ion monitoring; TPH = total petroleum hydrocarbons; TRPH = total recoverable petroleum hydrocarbons

determination is based on selected components or the sum of all components detected within a given range. Frequently the approach is to use two methods, one for the volatile range and another for the semivolatile range. Volatiles in water or solid samples are determined by purge-and-trap GC/FID. The analysis is often called the gasolines range organics (GRO) method. The semivolatile range is determined by analysis of an extract by GC/FID and is referred to as diesel range organics (DRO). Individual states have adopted methods for measuring GRO and DRO contamination in soil and water. The specific method details and requirements vary from state to state. Some of the GC TPH methods are summarized in Table 3-4.

In the mid-1980s underground storage tank (UST) programs were a focus of federal and state initiatives. The criteria and methodology for determining contamination are generally state-specific. Although many states still use EPA Method 418.1, GC procedures have been developed to provide more specific information on hydrocarbon content of waters and soils (Judge et al. 1997, 1998). GRO and DRO are specified in some cases, and several states, such as California and Wisconsin, aggressively developed programs to address groundwater contamination problems. These GC methods, coupled with specific extraction techniques, can provide information on product type by comparison of the chromatogram with standards. Quantitative estimates may be made for a boiling range or for a range of carbon numbers by summing peaks within a specific window. Although these methods provide more product information than the TPH and TOG methods, they are not without limitations. These include high results caused by interferences, low recovery due to the standard selected, petroleum product changes caused by volatility, and microbial activity (Restek 1994).

Many methods are available for analysis of petroleum hydrocarbon products, particularly in water and soil matrices. The current literature includes a number of studies that document the performance and limitations of the commonly used methods. Method modifications and new methods are being investigated to provide better information about the petroleum component content of environmental samples. However, the available analytical methodology alone may not provide adequate information for those who evaluate the movement of petroleum components in the environment or evaluate the health risks posed to humans (Heath et al. 1993a).

In its work to develop a fraction approach to assess TPH risks the TPH Criteria Working Group (TPHCWG) has developed an analytical method for identifying and quantifying the presence of the

Table 3-4. Summary of Gas Chromatographic TPH Methods

Method name	Matrix	Scope of method	Reference
Direct injection methods			
EPA Method 8015B	Solid wastes	Used to determine the concentration of petroleum hydrocarbons, including gasoline range organics (GROs). Analysts should use the fuel contaminating the site for quantitation.	EPA 1995c
ASTM Method D3328-90	Water	Petroleum oils such as distilate fuel, lubricating oil, and crude oil recovered from water or beaches. Identification of a recovered oil is determined by comparison with known oils, selected because of their possible relationship to the recovered oil.	ASTM 1994
Purge and trap and headspace methods			
EPA Method 8015B	Solid wastes	Used to determine the concentration of petroleum hydrocarbons, including diesel range organics (DROs) and jet fuel. Analysts should use the fuel contaminating the site for quantitation.	EPA 1995c

[a] Qualitative, screening procedur
DRO = diesel range organics; GRO = gasoline range organics; TPH = total petroleum hydrocarbons

groups or fractions with similar mobility in soils. The technique is based on EPA Method 3611 (Alumina Column Cleanup and Separation of Petroleum Wastes) and EPA Method 3630 (Silica Gel Cleanup), which are used to fractionate the hydrocarbon into aliphatic and aromatic fractions. A gas chromatograph equipped with a boiling point column (non-polar capillary column) is used to analyze whole soil samples as well as the aliphatic and aromatic fractions to resolve and quantify the fate-and-transport fractions selected by the TPHCWG (Gustafson 1997). The method is versatile and performance-based and, therefore, can be modified to accommodate data quality objectives (Gustafson 1997).

The Massachusetts Department of Environmental Protection (MADEP) approached its needs to evaluate the potential health effects of petroleum hydrocarbons similarly by defining analytical fractions. MADEP's method is based on standard EPA Methods (8020/8015 Modified), which allows it to be easily implemented by contract laboratories (Gustafson 1997; Hutcheson et al. 1996). Lighter hydrocarbon fractions (C_6-C_{12} are analyzed by purge-and-trap GC analysis using a FID to measure the total hydrocarbons and a photoionization detector (PID) to measure the aromatics. The aliphatic (e.g., hexane) component of the TPH is found by determining the difference. Aromatic and aliphatic fractions are divided into carbon number fractions based on the normal alkanes (e.g., *n*-octane) as markers. Heavier hydrocarbons (C_{12}-C_{26}) are analyzed using an extraction procedure followed by a column separation using silica gel (Modified EPA Method 3630) of the aromatic and aliphatic groupings or fractions. The two fractions are then analyzed using GC/FID. PAH markers and *n*-alkane markers are used to divide the heavier aromatic and aliphatic fractions by carbon number, respectively. A couple of concerns about the methodology have been expressed: (1) the PID is not completely selective for aromatics and can lead to an overestimate of the more mobile and toxic aromatic content; and (2) the results from the two analyses, purgeable and extractable hydrocarbons, can overlap in carbon number and cannot be simply added together to get a total TPH concentration.

Few methods are available for monitoring petroleum products in other matrices such as plant and animal tissue and food.

3.3.1.1 Soils and Sediments

Methods for determining TPH in soils and sediments are discussed in Section 3.3.1 above. These methods are used primarily for UST programs. Currently, many of the states have adopted EPA Method 418.1 or modified EPA Method 801.5 or similar methods for analysis during remediation of contaminated sites. Thus, there is no standard for TPH analysis; each state has adopted its own criteria, and in some cases, developed its own methodologies (Murray 1994).

There is a trend toward use of GC techniques in analysis of soils and sediments. One aspect of these methods is that "volatiles" and "semivolatiles" are determined separately. The volatile or GRO components are recovered using purge-and-trap or other stripping techniques (Chang et al. 1992; EPA 1995d; McDonald et al. 1984). Semivolatiles are separated from the solid matrix by solvent extraction (EPA 1995d). Other extraction techniques have been developed to reduce the hazards and the cost of solvent use and to automate the process (Gere et al. 1993). Techniques include supercritical fluid extraction (SFE) (Fitzpatrick and Tan 1993; Gere et al. 1993; Hawthorne et al. 1993; Lopez-Avila et al. 1993) microwave extraction (Hasty and Revesz 1995; Lopez-Avila et al. 1994) Soxhlet extraction (Martin 1992) sonication extraction (Martin 1992) and solid phase extraction (SPE) (Schrynemeeckers 1993). Capillary column techniques have largely replaced the use of packed columns for analysis, as they provide resolution of a greater number of hydrocarbon compounds.

3.3.1.2 Water and Waste Water

Methods for determining TPH in aqueous samples are discussed above in Section 3.3.1. The overall method includes sample collection and storage, extraction, and analysis steps. Sampling strategy is an important step in the overall process. Care must be taken to assure that the samples collected are representative of the environmental medium and that they are collected without contamination. There are numerous modifications of the EPA, American Public Health Association (APHA), and American Society for Testing and Materials (ASTM) methods discussed above. Most involve alternate extraction methods developed to improve overall method performance for TPH or replacement of the chlorofluorocarbon solvents. SPE techniques have been applied to water samples (Schrynemeeckers 1993). Solvent extraction methods with hexane (Murray and Lockhart 1981; Picer and Picer 1993) or methylene chloride (Mushrush et al. 1994) have been reported as well.

3.3.1.3 Air

Methods for determining hydrocarbons in air matrices usually depend upon adsorption of TPH components onto a solid sorbent, subsequent desorption and determination by GC techniques. Hydrocarbons within a specific boiling range (*n*-pentane through *n*-octane) in occupational air are collected on a sorbent tube, desorbed with solvent, and determined using GC/FID (NIOSH 1994). Although method precision and accuracy are good, performance is reduced at high humidity.

Compounds in the boiling range 80-200 ºC in ambient air may be captured on a Tenax GC adsorbent tube which is thermally desorbed for GC/MS analysis (EPA 1988). Performance of the method had not been established on a compound-by-compound basis (EPA 1988). Gasoline vapor in air may be sampled on a tube containing Tenax adsorbent. The traps are thermally desorbed and analyzed by GC/FID. The minimum detectable concentration is 0.03 mg/m^3 total hydrocarbons in a 2.5 L sample. Excellent recovery was reported (>90%) (CONCAWE 1986). Passive adsorbent monitors (badges) may also be used. Compounds are solvent-desorbed from the exposed adsorbent and analyzed by GC. Good recovery (>80%) has been reported for target *n*-alkanes and for gasoline, naphtha, and Stoddard solvent (3M 1993).

The Massachusetts Department of Environmental Protection (MADEP), along with ENRS, Inc., of Acton, Massachusetts, has developed a method for taking and analyzing air samples for the presence of petroleum hydrocarbons (MADEP 1999). This Air-phase Petroleum Hydrocarbon (APH) method uses SUMMA canisters and GC/MS for sampling and analysis of ambient air, indoor air, and soil gas. This method can be downloaded from the MADEP website (http://www.state.ma.us/dep). The complex mixture of petroleum hydrocarbons potentially present in an air sample is separated into aliphatic and aromatic fractions, and then these two major fractions are separated into smaller fractions based on carbon number. Individual compounds (e.g., benzene, toluene, ethylbenzene, xylenes, MTBE, naphthalene) are also identified using this method. The range of compounds that can be identified includes C4 (1,3-butadiene) through C 12 (*n*-dodecane).

Continuous monitoring systems for total hydrocarbons in ambient air are available. These usually involve flame ionization detection. Detection limits are approximately 0.16 ppm (Lodge 1988).

3.3.2 Biological Samples

Few analytical methods were located for determination of TPH in biological samples. However, analytical methods for several important hydrocarbon components of total petroleum hydrocarbons may be found in the ATSDR toxicological profiles listed in Table 3-1.

Some methods developed for analysis of aquatic and terrestrial life may be adaptable to human biological samples. Examples are summarized in Table 3-5. Most involve solvent extraction and saponification of lipids, followed by separation into aliphatic and aromatic fractions on adsorption columns. Hydrocarbon groups or target compounds are determined by GC/FID or GC/MS. These methods may not be suitable for all applications, so the analyst must verify the method performance prior to use.

Methods are also available for determination of specific hydrocarbon compounds in biological samples. Some of these methods are shown in Table 3-5. Since these methods have not been demon- strated for total petroleum hydrocarbons, the analyst must verify that they are suitable prior to use.

3.3.3 Adequacy of the Database

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of TPH is available. Where adequate information is not available, ATSDR, in conjunction with the NTP, is required to assure the initiation of a program of research to determine the health effects (and techniques for developing methods to determine the health effects) of TPH. Since TPH is comprised of a number of component chemicals, these directives and requirements can be assumed to extend to the individual compounds that may be found as components of TPH.

Health assessment of the risks associated with petroleum hydrocarbons from environmental media are difficult because of the complex nature of petroleum products, lack of adequate knowledge about the movement of petroleum components in soil, and lack of knowledge about the toxicity of the components (Heath et al. 1993a). Health assessors often select surrogate or reference compounds (or combinations of compounds) to represent TPH so that toxicity and environmental fate can be

Table 3-5. Analytical Methods for Determining Total Petroleum Hydrocarbons in Biological Samples

Sample matrix	Preparation method	Analytical method	Sample detection limit	Percent recovery	Reference
Human milk (purgeable hydrocarbons)	Purge and trap; thermal desorption	Capillary GC/MS	Not reported	35–88[a]	Pellizzari et al. 1982
Blood	Heated headspace extraction	Capillary GC/MS-SIM	50 picograms injected	No data	Kimura et al. 1991
Blood	Purge and trap	Isotope dilution: capillary GC/high resolution MS	Low ppt	91–147	Ashley et al. 1992
Breath	Collection in passivated cannisters using spirometer system	Capillary GC/MS	Low $\mu g/m^3$ range	25–136	Thomas et al. 1991

[a] Data for 4 control compounds

FID = flame ionization detector; GC = gas chromatography; MS = mass spectrometry; TPH = total petroleum hydrocarbons

evaluated. One approach is based on benzene as the most appropriate substitute for TPH based on its toxicity, motility in the environment, and solubility in ground water (Youngren et al. 1994). Other researchers have investigated the use of several surrogate compounds to represent the movement of TPH in the environment and TPH toxicity. Potential candidates are *n*-hexane, benzo(a)pyrene, and pyrene to represent alkanes, carcinogenic PAHs, and noncarcinogenic PAHs in gasoline, respectively. Benzene and toluene would be included for sites where the BTEX portion of gasoline is not analyzed separately (Koblis et al. 1993).

Another approach is to categorize hydrocarbon compounds into surrogate fractions characterized by similar chemical and physical properties (EA Engineering 1995). Compounds are assigned to a given fraction on the basis of similar leaching and volatilization factors. Correlation to Carbon Number Index was used because it closely follows GC behavior. This method has the potential to provide realistic evaluation of potential risks; however, a full set of parameters is not available for all the compounds of interest (EA Engineering 1995).

3.3.4 Ongoing Studies

Governmental, industrial, and environmental groups have been attempting to understand the problems of environmental contamination with petroleum hydrocarbons. Major agencies, such as the International Agency for Research on Cancer (IARC) and the EPA are involved in the discussion of potential health effects. Some groups have been attempting to improve the analytical consistency and interpretation of results in dealing with petroleum hydrocarbons, and some have looked at the health and environmental effects of petroleum. The ASTM publishes consensus standards, including analytical methods. Committee D-19 of ASTM is concerned with the study of water and is responsible for the standardization of methods for sampling and analysis of water, aqueous wastes, water-formed deposits, and sediments. Committee D-2 on Petroleum Products and Lubricants is responsible for the ASTM *Manual on Hydrocarbon Analysis* (ASTM 1992).

The Amherst annual conference continues to address issues surrounding petroleum contamination, including analytical methods (Amherst 1999). Though the TPHCWG has taken on specific responsibilities for TPH, further analytical developments will likely grow from this conference.

In another ongoing effort, EPA is looking at the problem of petroleum wastes in all media. They have formed an internal working group and are supporting the efforts of other groups such as the Amherst Conference Workgroups and Workshop on General Population Exposures to Gasoline (Lioy 1992).

Dr. R.J. Rando, Tulane University, is investigating the use of passive samplers for measuring hydrocarbon components. The overall goal of the program is to characterize and improve the performance of passive samplers for use in ambient and indoor air monitoring.

Petroleum companies have conducted a number of studies regarding the health effects of TPH constituents and products that have not appeared in the open published literature (API 1995a).

4. PRODUCTION, IMPORT/EXPORT, USE, AND DISPOSAL

This chapter summarizes useful background materials dealing with petroleum production and a range of common products derived from petroleum contaminants that could be documented through TPH testing at NPL sites. The chapter concludes with a discussion of acceptable disposal practices for petroleum products. In conjunction with materials in Chapter 5, the section on disposal summarizes special features of petroleum that set it apart from a variety of more highly processed petrochemicals. Under normal uses as fuels, lubricants, or paving materials, petroleum products are not considered hazardous materials. For instance, fuels are normally consumed through combustion processes to drive motors or provide space heating. Some combustion by-products (e.g., carbon monoxide) may be regarded as hazardous, but a variety of legal exemptions apply to the initial petroleum product, at least under federal law.

The special status of petroleum under normal use means that limited attention is given to monitoring of petroleum levels in the workplace or the environment. It is usually only in the case of accidental spills, pipeline breaks, or seepage from storage tanks that well defined legal requirements are in place that require record keeping and documentation. As a result, it is usually hard to make precise connections between the original petroleum products and the types of TPH materials encountered at NPL sites.

Especially at older dump sites, original petroleum product mixtures become even more complex mixtures. Over time, biotic and abiotic weathering processes alter the types of chemical fractions still present on-site. This means that even the most detailed knowledge of the various original petroleum products does not necessarily provide clear signals on the exposure risks affecting an NPL site with TPH contaminants. See Chapter 5 for discussion of environmental transport and potential human exposure. This chapter, therefore, highlights basic information relevant to the original petroleum products to provide a background for the discussion on environmental fate and transport issues in the next chapter.

Background on Primary Petroleum Products. Petroleum is a natural resource found in many types of sedimentary rock formations. Naturally occurring petroleum is a complex mixture of gaseous, liquid, and solid hydrocarbons. Entire industries have grown up around the activities

required to produce the crude oil, transport it to refineries, and convert the natural petroleum into a variety of end products and chemical feedstocks. Processed petroleum products provide up to 50% of the world's total energy supply, major forms of transportation, electric utilities, and space heating. Petroleum is also used in lubricants, solvents, highway surfacing, and roofing and waterproofing materials, and as the source of the feedstocks used to make plastics and other modern petrochemicals.

Early refining techniques relied primarily on the separation of different fractions from the raw petroleum using distillation over different temperature ranges. For straight-chain, branched, and aromatic hydrocarbons, there is some degree of correlation between the number of carbon atoms in a compound and the boiling point. Many refined products were initially given simple technical definitions based on the temperature range at which a certain fraction was extracted from the crude oil. The very lightest fractions (e.g., C_4H_{10} or butane and other simple straight-chain compounds down through CH_4 or methane) were traditionally vented or flared since there was little apparent demand for these gaseous components. The most prized fractions were liquids at normal room temperatures that could be used as fuels in engines or as heating oils.

The petroleum refining industry has tried to find profitable uses for both the lighter and heavier crude oil fractions. Lighter gaseous fractions can now be used for space heating or fuels in the form of liquified petroleum gas (LPG). For the heavier fractions, a variety of technologies convert large hydrocarbon molecules from the distilled crude oil into lighter compounds that can be used as motor gasoline, aviation fuel, or fuel oil. In the process, large amounts of hydrocarbons are produced that can be isolated as relatively pure substances for use as solvents or petrochemical feedstocks. For instance, benzene was once derived from coal tars, but most supplies are now derived from oil. Ethane is easily converted into ethylene, a major petrochemical feedstock. Commercial techniques for producing xylenes, toluene, butadiene, butylenes, and propylene also involve simple adaptations of modern oil refinery technologies.

Some specific refinery-generated hydrocarbons are blended into gasolines or fuel oils to enhance some desired property. For example, commercially pure grades of toluene and benzene are added to modern gasoline to boost octane ratings. Similar enhancements in basic product qualities for combustion or viscosity are achieved through re-distilling products from the cracking process and blending them with fractions obtained from primary distillation. While the resulting products are still

referred to as gasolines or fuel oils, the chemistry of the hydrocarbons in these mixtures often differs considerably from that of the hydrocarbons found in the original crude oil.

Refining also dramatically increases the frequency of hydrocarbons in which carbon-hydrogen bonds have been replaced with double bonds between carbon atoms. The resultant chemicals are called olefins and include ethylene (C_2H_4), propylene (C_3H_6), and butylene (C_4H_8). While the lighter forms such as ethylene are relatively easy to remove for use as petrochemical feedstocks, a variety of heavier olefins wind up in the refinery gasoline or fuel oil products.

In addition to aromatics with benzene ring structures, modern refinery processes tend to increase the number of hydrocarbons with simpler types of carbon ring structures. Typical chemicals include cyclopentane, where the straight-chain pentane has been wrapped into a five-carbon ring. Other transformations of aliphatic hydrocarbons include cylcohexane and cyclopentane. These ring compounds are usually called naphthenes.

These complex alterations in the types of compounds generated from refinery operations have led to the development of a variety of technical nomenclatures to describe different petroleum fractions. Many commercial products still carry such traditional names as gasoline or heating oil. In terms of such basic physical and chemical properties as specific gravities and combustion performance, these traditional labels have held their meanings fairly well. New products, such as fuel oils derived from residuals, now join the original fuel oils derived from simple distillation, but the term “fuel oil” is still commonly used to organize data on petroleum imports, exports, and production. But the chemistry of these modern products is often considerably more complex than the chemistry of pre-World War II products with the same names.

Petroleum Production, Import/Export, and Use in the United States.

Petroleum Production and Use Statistics. Petroleum use and production statistics pooled from a variety of government and industry sources are available from the PennWell Publishing Company. A convenient printed compendium (also available on computer disk in a digitized form) is the *Energy Statistics Sourcebook* (PennWell 1994).

During 1997, total U.S. crude oil production was 2,300,000,000 barrels (API 1998a). Using consistent estimation methods comparable to those employed over the last decade, it is often difficult to match current petroleum product statistics with historical statistics developed prior to 1978. For 1978, total U.S. crude production was 3,178,216,000 barrels. This represents a 27.6% decline in total production between 1978 and 1998. While total crude oil production in the United States has shown an overall downward trend, a comparison of statistics from 1993 and 1978 indicates that the total output from refineries based in the United States has remained remarkably constant. Table 4-l summarizes total refinery output along with output estimates for major refinery petroleum products. Output for specific refinery products has changed: jet fuel kerosenes and LPG have increased, and fuel oils recovered from heavier refinery residuals and ordinary kerosene have decreased. Crude oil production levels and trends for selected states are summarized in Table 4-2.

Statistics on crude oil production or its processing into various petroleum fractions are generally presented using a standard barrel (42 U.S. gallons) as the basis of comparison. The barrel is still an international standard for crude oil statistics. While adjustments can be made for particular types of crude oil related to variations in their specific gravities (e.g., light oils versus heavy oils), 7.3 barrels of crude oil equal approximately 1 metric ton (1,000 kg or 2,204.6 pounds). Conversion factors are also available to make estimates of the barrel equivalents of other common petroleum products ranging from to liquified petroleum gas (LPG). Conversion factors for major petroleum fractions are given in Table 4-3.

Although crude oil production is the source of TPH exposures to certain occupational groups and people living near oil production sites, the releases in workplaces or to environmental media of more concern for this profile begin during the stage when crude oil is refined and transformed into a variety of petroleum products for fuels, lubricants, and petrochemical feedstocks.

In addition to the total production figures, percentage breakouts provide another way to summarize the major products stemming from U.S. based refineries. Table 4-4 presents 1993 product yields on a percentage basis.

Table 4-1. U.S. Annual Refinery Output (in 1,000s of Barrels)

Product	1978 Annual output	1998 Annual output
Total production	5,825,041	1,969,729
Motor gasoline	2,616,656	906,459
Jet naphtha	65,257	73
Jet kerosene	288,682	87,112
Kerosene	56,325	8,894
Distillate fuel oil	1,156,097	403,597
Residual fuel oil	608,634	92,639
Liquid petroleum gas and lighter fractions	129,526	79,388

Source: PennWell 1998

Table 4-2. Crude Oil Production Trends by State

State trends	1978 Production 1,000s of barrels	1997 Production 1,000s of barrels	Percent change where more than 10%
Texas	1,074,050	594,103	-44.69
Alabama	19,829	14,831	-25.21
Alaska	448,620	472,949	28.73
Montana	30,467	15,527	-49.04
Arkansas	20,329	8,429	-58.54
Nebraska	5,862	3,337	-43.07
Utah	31,368	19,317	-38.42
Virginia	2	10	400
Arizona	418	82	-80.38
South Dakota	869	1,334	53.51
North Dakota	24,812	35,833	44.42
West Virginia	2,382	1,508	-36.69
Missouri	54	114	111.11
Pennsylvania	2,887	1,320	-54.28
New York	852	276	-67.61
Louisiana	532,740	488,784	-21.64
Florida	47,536	6,381	-86.58
Indiana	4,689	2,430	-48.18
Nevada	1,156	980	-15.22
Tennessee	593	367	-38.11
Kansas	56,586	39,836	-29.60
Kentucky	5,724	2,988	-47.8
Mississippi	42,024	21,037	-49.94
Colorado	36,797	25,616	-30.39
Michigan	34,667	10,052	-71.00
Illinois	23,362	16,115	-31.02
Oklahoma	150,456	83,365	-44.59
Ohio	11,154	8,593	-22.96
California	347,181	339,306	
Wyoming	137,385	70,176	-48.92
New Mexico	83,365	69,835	-16.23

Source: PennWell 1998

Table 4-3. Barrel Oil Equivalents of Petroleum Liquid Fuels

Fuel	Thousands of barrels of oil equivalent	
	Per thousand metric tons	Per million U.S. gallons
LPG	11.6	23.8
Aviation gasoline	8.9	23.8
Motor gasoline	8.5	23.8
Jet fuel (gasoline types)	8.3	23.8
Naphtha	8.5	23.8
Kerosene	7.8	23.8
Jet fuel (kerosene types)	7.7	23.8
Distillate fuel oil	7.3	23.8
Residual fuel oil	6.7	23.8
Lubricating oil	7.1	23.8
Typical crude oil	7.3	23.8

Source: Stevens 1988

Table 4-4. 1997 U.S. Refinery Output of Major Products as Percentages of Total Refinery Output

Product	Percentage of total output
Asphalt and road oil	3.2
Distillate fuel oil	22.5
Jet fuels	10.3
Kerosene	0.4
LPG and Ethane	4.6
Lubricants	1.2
Motor gasoline	45.7
Petrochemical feedstocks	2.9
Petroleum coke	4.6
Residual fuel oil	4.7
Other products (approximately)	5.0

Source: PennWell 1998

With a long-term decline in the levels of domestic crude oil production, imports have increased to meet the demand for petroleum products and to sustain the fairly stable levels of U.S.-based refinery output. Tables 4-5, 4-6, and 4-7 summarize trends in petroleum product imports, exports, and levels of U.S. demand (use) for these products.

For the most common refinery products, statistics are available showing U.S. use patterns for sectors such as major industrial groups or residential demand. These statistics are presented in Table 4-8.

Disposal. An estimated 2.3 billion barrels of crude oil were produced in 1997 (API 1998a). From this crude oil, TPH waste may be generated in a number of ways that ultimately lead to either improper or acceptable disposal. Incineration is a primary method of disposal for wastes containing TPH. Oil spills are frequently captured and treated using various absorbents (e.g., straw, polyurethane foam, activated carbon, peat), gelling agents, dispersants, and mechanical systems. Biodegradation also has been used to treat contaminated soil (OHM/TADS 1985).

Sources of TPH waste include

- waste generated from crude oil production,
- waste generated from petroleum refining,
- used oil as a waste,
- used petroleum refining products as wastes, and
- accidental releases of crude oil, petroleum refining wastes, used oil, and petroleum refining products.

Management of TPH wastes generated from the sources listed is discussed in the following sections, which address existing regulatory programs, quantities disposed (where data are available), waste management trends, recycling trends, and records of damage for each source.

Waste Generated from Crude Oil Production. EPA's Report to Congress, *Management of Wastes from the Exploration, Development, and Production of Crude Oil, Natural Gas, and Geothermal Energy* (EPA 1987a), reported that the American Petroleum Institute estimated that 361 million barrels of waste were generated from the drilling of 69,734 oil wells in 1985. This translates into about 5,183 barrels of waste per well. These wastes are not pure crude but can include petroleum hydrocarbons.

Table 4-5. U.S. Petroleum Imports for 1978 and 1998

Product	1978 (1,000s Barrels)	1998 (1,000s Barrels)
Crude oil	2,261,026	974,667
All refined products	732,819	212,625
Motor gasoline	69,518	32,989
Jet naphtha	6,963	0
Jet kerosene	24,383	9,606
Kerosene	4,031	190
Distillate fuel oil	63,288	23,428
Residual fuel oil	494,640	24,331
LPG and ethane	44,827	26,988

Source: PennWell 1998

Table 4-6. U.S. Petroleum Exports for 1978 and 1998

Product	1978 (1,000s Barrels)	1998 (1,000s Barrels)
Crude oil	57,728	20,621
All refined products	74,329	99,106
Motor gasoline	470	13,618
Jet naphtha	1	232
Jet kerosene	513	3,703
Kerosene	40	99
Distillate fuel oil	1,202	15,905
Residual fuel oil	4,634	16,624
LPG and ethane	7,238	5,562

Source: PennWell 1998

Table 4-7. U.S. Petroleum Demand for 1978 and 1998

Product	1978 (1,000s Barrels)	1998 (1,000s Barrels)
Motor gasoline	2,705,309	943,156
Jet naphtha	72,546	-183
Jet kerosene	313,108	187,359
Kerosene	64,042	11,634
Distillate fuel oil	1,252,556	424,436
Residual fuel oil	1,103,233	101,591
LPG and ethane	511,598	256,941

Source: PennWell 1998

Table 4-8. Petroleum Use Patterns by Sector for 1995

Sectoral use patterns for major petroleum products
1995 baseline
Millions of barrels (percent of total sectoral demand)

Product	Residential	Commercial	Industrial	Transportation	Electric utilities	Total
Motor gasoline	0 (0.0)	3 (<1.0)	38 (1.3)	2,801 (98.6)	0 (0.0)	2,842
Kerosene	13 (65.0)	4 (20.0)	3 (15.0)	0 (0.0)	0 (0.0)	20
Distillate fuel oil	152 (13.0)	79 (6.8)	184 (15.7)	740 (63.2)	16 (1.4)	1,170
Residual fuel oil	0 (0.0)	23 (7.4)	54 (19.9)	147 (47.3)	87 (28.0)	311
LPG and ethane	112 (16.2)	20 (2.9)	557 (80.4)	5 (0.7)	0 (0.0)	693

Source: PennWell 1998

Wastes include drilling fluids and produced waters which are managed in pits, discharged to surface waters, or injected into the producing well or an aquifer (Charbeneau et al. 1995). Records of damage due to both improper and acceptable management of these wastes reflects the presence of constituents of concern found in crude oil such as benzene, phenanthrene, lead, and barium. Numerous damage cases are cited in this Report to Congress, including an estimated 425 reported spills on the North Slope of Alaska in 1986.

Current regulatory programs applicable to these wastes include a variety of state programs, the Underground Injection Control Program established under the Safe Drinking Water Act Part C (Class II wells are oil and gas-related), and the Bureau of Land Management regulations for the activities on federal and Indian lands.

Wastes Generated from Petroleum Refining. Petroleum refining wastes are regulated by EPA in several ways. There are approximately 150 active petroleum refineries in the United States. RCRA Subtitle C currently lists four characteristics as hazardous in 40 CFR 264.21 and .24 and five waste categories as hazardous in 40 CFR 261.31 and .32. When most of these wastes were listed beginning in 1980, there were 250-300 active refineries ranging in capacity from about 400,000 barrels (bbl) per day to only a few hundred bbl per day.

In addition, petroleum refining wastes are subject to evaluation as characteristically hazardous waste, including the toxicity characteristic (40 CFR 261, Subpart C) which labels wastes “RCRA hazardous” if a measured constituent concentration exceeds a designated maximum (e.g., a benzene concentration of 0.5 mg/L)

All Subtitle C hazardous wastes are prohibited from land disposal without prior demonstration that hazardous constituent concentration levels comply with regulatory limits or that prescribed methods of treatment are used. These two criteria are intended to reduce the toxicity of the waste or-substantially reduce the likelihood of migration of hazardous constituents from the waste, so that health and environmental threats are minimized. The primary method of treatment is waste combustion to destroy organic constituents.

RCRA-classified listed hazardous wastes are also hazardous substances under the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA), as amended. CERCLA hazardous substances are listed in 40 CFR 302.4 and have unique reportable quantities (RQs) which, when released, trigger emergency response and reporting measures.

Oil generated and recovered during petroleum refining has also been excluded from RCRA regulation. In 1994, EPA limited the exclusion to recovered oil from refining, exploration, and production that is inserted into the petroleum refining process prior to distillation and catalytic cracking. Recovered oil includes materials that are primarily oil and that are recovered from any phase of petroleum exploration, refining production, and transportation. It is considered by EPA to be equivalent to the raw materials normally used in refining in composition and management. In November 1995, EPA proposed to expand this exclusion to encompass all oil-bearing secondary materials that are generated within the petroleum refining industry and that are reinserted into the refining process (including distillation, cracking, fractionation, or thermal cracking).

Used Oil as a Waste. "Used oil means any oil that has been refined from crude oil, that has been used and as a result of such use is contaminated by physical or chemical impurities" (40 CFR 260.10). In 1992, there were approximately 700,000 commercial, industrial, and large farm used oil generators in the United States. The management of used oil has a statutory, regulatory, and judicial history dating back to 1978. Currently, used oil exhibiting any hazardous waste characteristics must be managed under RCRA Subtitle C as a hazardous waste. In turn, used oils contaminated with CERCLA hazardous substances are subject to RQs under 40 CFR 302.4. Disposal of nonhazardous used oil that is not recycled is regulated under 40 CFR 257 and 258 of RCRA Subtitle D. The recycling of all used oils is regulated under 40 CFR 279. These regulations include programs for generators, collection centers, transporters and transfer facilities, processors and re-refiners, burners, and marketers. An estimated 750 million gallons per year of used oil enter the commercial used oil recycling system according to EPA. In 1992, these recycling businesses consisted of independent collectors (383), minor processors (70), major processors (112), re-refiners (4), fuel oil dealers (25-100) and burners (1,155). Products of used oil processing and re-refining include specification fuel, reconstituted lubricating oils and fluids, distillate fuel, lube feedstock, asphaltic bottoms, and other non-fuel oil-derived products. Part 279 prohibits used oil use as a dust suppressant unless a state successfully petitions for authority to allow its use as a suppressant. As of 1992, 41 of 50

states prohibited road oiling. No regulations exist for individuals who generate used oil through home or personal use of oil products.

Used Petroleum Refining Products as Wastes. Government regulations presume that petroleum refining products are consumed and not disposed. Therefore, there are no regulatory programs designed for the intentional disposal of petroleum products. However, RCRA can apply to disposed petroleum products. These products can be declared solid wastes and, possibly, hazardous waste as defined under 40 CFR 261. The only exemption from the definition of solid waste for petroleum products is when the material is recycled. There are no exemptions from the definition of hazardous waste for petroleum products declared to be wastes. Used oil, in particular, has a specific RCRA regulatory program, as described above.

Petroleum products such as gasoline contain certain hazardous constituents including benzene, toluene, and xylene. However, the presence of such constituents in gasoline does not qualify it as a hazardous waste under RCRA or a hazardous substance under CERCLA. The management of petroleum products is, however, regulated under three programs: Underground Storage Tanks (UST) (40 CFR Part 280) to prevent tank leakage, Hazardous Materials Transportation (HMT) (49 CFR Chapter 1) for petroleum distillates with combustible and flammable properties, and the Occupational Safety and Health standards (29 CFR Part 1910.1000) for inhalation hazard. The UST and HMT programs are designed to prevent and respond to accidental releases of petroleum products. Both programs are discussed in the next section.

Accidental Releases of Crude Oil, Petroleum Refining Wastes, Used Oil, and Petroleum Refining Products

Oil Production Wastes. Numerous damage cases are cited in the 1987 EPA Report to Congress, including the estimated 425 reported spills on the North Slope of Alaska in 1986. However, EPA did not believe the impact of these releases warranted regulating these oil production wastes as RCRA hazardous. Rather, they are regulated under state programs.

Petroleum Refining Waste. The extent of mismanagement or accidental releases of petroleum refining wastes can be illustrated with the 1995 proposed RCRA listing determination for 16

additional petroleum refining waste categories (of which 3 waste categories were determined to be RCRA hazardous and proposed to be listed in 40 CFR 261). A search of state and federal enforcement records, documented CERCLA-related activities at 10 sites and RCRA-related activities at 29 sites.

Accidental releases of RCRA-listed petroleum refining wastes are regulated in two ways. First, as part of the RCRA program, treatment storage and disposal facilities (TSDFs) that manage hazardous refinery wastes must obtain permits. A key component of the permit application is demonstration of an effective contingency plan for accidental releases, a preparedness/prevention plan, and a groundwater monitoring plan when wastes are managed in land-based units among other activities. Second, these wastes are also subject to the reportable quantity (RQ) requirements of CERCLA.

Used Oil and Other Waste Petroleum Refining Products. Used oil and other petroleum product mismanagement and related risks are controlled under other regulations and statutes; these include the 40 CFR Part 268 underground storage tank (UST) regulations, the 40 CFR Part 112 Spill Prevention, Control and Countermeasure (SPCC) program, the National Pollutant Discharge Elimination System (NPDES) storm water regulations, and the lead phase-down program. Section 311 of the Clean Water Act requires facilities to have an SPCC plan or contingency plan in place to ensure that oil spills are prevented, controlled via containment measures, and responded to when oil spills occur and reach navigable waterways. About 50% or more of the used oil generators, and most of the used oil transporters, processors/re-refiners, and off-spec used oil burners are covered by the SPCC program. Less than 10% of the used oil industry participants are excluded from the SPCC program because they are not in the vicinity of navigable waterways. The program includes non-transportation-related facilities located in proximity to navigable waters, USTs with capacities greater than 42,000 gallons, aboveground storage tanks with capacities greater than 1,320 gallons, and single tanks with capacities greater than 660 gallons.

The International Convention for the Prevention of Pollution from Ships (1973) as modified by the 1978 Protocol (MARPOL) focuses on preventing ship-generated ocean pollution. Annexes I-V of the MARPOL protocol address oil, noxious liquids, and other petroleum-related contaminants (MARPOL 1978).

The Hazardous Materials Transportation Act regulates used oil and petroleum distillates if they meet the definitions of “flammable” or “combustible.” All used oil generators and transporters must comply with applicable Department of Transportation regulations for hazardous materials (49 CFR, Chapter I - Research and Special Programs Administration).

The Toxic Substances Control Act (TSCA) prohibits the use of waste oil containing any detectable polychlorinated biphenyls (PCBs) as a sealant, coating or dust suppressant. Any spill of material containing ≥50 ppm PCBs into the sewer, drinking water, surface water, grazing land, or vegetable gardens must be reported (40 CFR 761).

The UST program (40 CFR Part 280) focuses on control and prevention of petroleum leaks from underground petroleum storage tanks including petroleum products and waste oil tanks. The regulations currently exempt UST systems less than 110 gallons in capacity, machinery containing substances regulated under the UST program, farm or residential tanks less than 1,100 gallons in capacity, heating oil tanks where the heating oil is used on the premises, and flow-through process tanks, among others.

5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

Petroleum products are an integral part of our modern lives. It is nearly impossible to avoid exposure to hydrocarbons from petroleum products, whether it is from gasoline fumes at the pump, spilled crankcase oil on asphalt, solvents used at home or work, or pesticide applications that use petroleum products as carriers. There are concerns with both short-term (accidents) and long-term exposures to petroleum hydrocarbons (e.g., contaminated drinking water). Gross measures of Total Petroleum Hydrocarbons (TPH) in soil or water are not particularly valuable for assessing either the potential for exposure to TPH or the impacts of such exposure on public health. This chapter addresses questions related to the first point: what are the levels of contamination in the environment, what happens to petroleum hydrocarbons in the environment, and what is the likelihood that individuals or populations will be exposed to petroleum hydrocarbons at levels thought to be of concern?

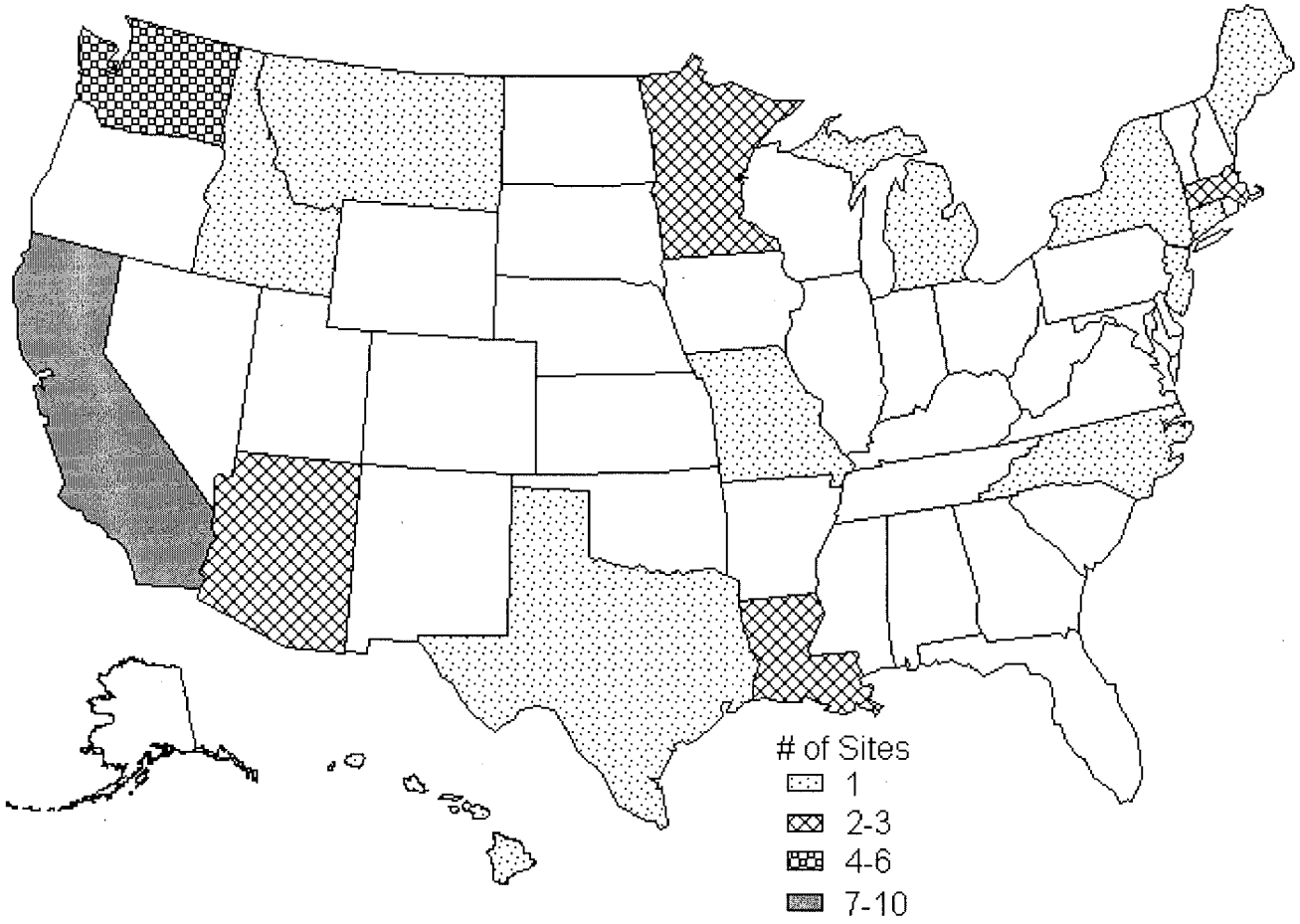
Petroleum products are released to the environment through accidents, as managed releases, or as unintended by-products of industrial, commercial or private actions. An understanding of the changes that occur over time in the composition of petroleum hydrocarbons found in soil, water, or air is extremely important in addressing public health issues for TPH. The TPH Criteria Working Group (TPHCWG 1997b) has defined its TPH fractions by the mobility of constituents in order to address this question of predicting risks associated with TPH contamination.

The following sections present an overview of releases to the environment (5.2), fate and transport (5.3), and levels found in the environment (5.4).

5.2 RELEASES TO THE ENVIRONMENT

TPH has been identified in 34 of the 1,519 current or former EPA National Priorities List (NPL) hazardous waste sites (ATSDR 1998a). Components of TPH are common environmental contaminants in all media and are likely contaminants at many NPL sites. However, the number of sites evaluated for TPH and TPH components is not known. The frequency of the TPH reported sites within the United States can be seen in Figure 5- 1.

Figure 5-1. State Density of NPL Sites with TPH Contamination



Source: HazDat 1999

The number of NPL sites reporting TPH contamination is a small subset of those contaminated with petroleum hydrocarbons.

Raw petroleum and refined petroleum products used as fuels or lubricants are generally excluded at the national level from the cradle-to-grave record-keeping associated with recognized toxics such as heavy metals or chlorinated solvents. With an eye to the availability of petroleum as a source of energy, petroleum production is tracked by the federal government as well as industry trade associations. Statistics are available for wellhead production as well as for production of major bulk fuel types from domestic refineries. These primary production statistics have been summarized in Chapter 4.

Once processed into products such as motor gasoline and fuel oil, most of the petroleum is burned in engines or boilers to provide energy for transportation, space heating, or electricity. In these combustion processes, the petroleum fuel is oxidized. Because of incomplete oxidation, small amounts of hydrocarbon emissions result. These emissions often contain much larger percentages of combustion by-products such as polycyclic aromatic hydrocarbons (PAHs) than the initial petroleum products. Incomplete combustion and heat also alter the composition of crank case oils and lubricants.

Emissions statistics are usually lacking for TPH or most TPH fractions since there is no record-keeping associated with smaller internal combustion engines used in cars and trucks or fuel oil boilers for individual buildings or homes. These individual uses account for the majority of petroleum product use. These releases, mostly to the atmosphere from incomplete combustion, however, are generally small compared to a variety of other releases connected with spills or uncontrollable losses during storage, transport, or fueling operations.

The movement of raw petroleum to automobile fuel tanks or fuel oil boilers is part of a complex bulk product distribution and storage system, providing many opportunities for accidents, spills, leaks, and losses from simple volatilization. Consistent national statistics are lacking for many stages in the overall oil distribution and storage system. The main exceptions involve larger leaks and spills, especially spills in coastal areas or on larger navigable rivers.

Data for the period from 1984 through 1993 (API 1996) show that most data reported to the U.S. Coast Guard occurred in inland bodies of water: rivers, lakes, and points on bays or estuaries. Spills from large ocean-going tankers and large spills in general (more than 1,000 gallons) are relatively

infrequent, never more than 5% of the total number of reported spills in a year. The average number of spills during the 1984-93 period was just under 6,000 spills. The numbers in any given year can vary enormously, with a maximum of just under 9,600 spills reported in 1991.

In terms of the amounts of oil estimated from spills, large spills, although rare, can dominate the annual totals. For instance, of about 14,000,000 total estimated gallons of oil spilled to U.S. waters in 1989, 10.8 million gallons resulted from the Exxon Valdez catastrophe in the coastal waters of Alaska. One or two large tanker spills in the course of a decade can make it very hard to draw conclusions on trends. The average amount of oil spilled in the 5-year period from 1984 to 1988 was 6.3 million gallons per year, compared to 5.6 million gallons spilled from 1989 to 1993. With eventual implementation of double-hull requirements for large tankers required in the Oil Pollution Act of 1990, the releases from tankers should be greatly curtailed.

Within the broad reporting categories of vessels (tankers and barges) and facilities (pipelines, tanks batteries, and other onshore facilities) in the period 1984-1993, numbers of reported spill incidents were roughly equivalent: 42,000 incidents from vessels and 38,000 from facilities. Over this period, the vessels spilled a much larger cumulative amount of oil: 45 million gallons from vessels versus 15 million gallons for facilities. Major incidents can dominate these totals. Two vessel spills account for around one-third of the vessel totals.

Most spills involve either crude oil or bulk fuels (distillates) such as fuel oils. Four tables (adapted from API 1996) help summarize annual figures on oil spills to coastal and inland waters of the United States. Table 5-l shows statistics on the number of spills broken out by size categories, where the prevalence of very small releases is obvious. Table 5-2 summarizes releases from vessels, and Table 5-3 summarizes releases from facilities. Table 5-4 summarizes spills according to the type of petroleum product involved.

At the national level, virtually the only other regulatory program that provides broad-based statistics on petroleum product releases to the environment is EPA’s (leaking) Underground Storage Tank (UST) Program. In 1994, there were over a million underground storage tanks on more than 300,000 identified UST sites; about 91% of these involve tanks at gasoline stations, truck stops, vehicle repair shops, or convenience stores selling gasoline or diesel fuel (EPA 1998c). There were at least

Table 5-1. Total Number of Oil Spills by Size: 1984–1996

Year	Under 10 Gallons	10–999 Gallons	1,000–9,999 Gallons	10,000–99,999 Gallons	More than 100,000 Gallons	Total
1984	5,446	3,273	366	74	16	9,175
1985	4,210	2,571	311	52	9	7,153
1986	3,737	2,285	128	25	11	6,186
1987	3,544	2,250	128	23	8	5,953
1988	3,626	2,238	125	29	11	6,029
1989	5,024	2,580	136	37	7	7,784
1990	6,480	2,720	164	43	11	9,418
1991	6,791	2,620	138	25	3	9,577
1992	6,322	2,556	105	22	3	9,008
1993	6,897	2,316	120	16	3	9,352
1994	6,659	2,376	144	21	8	9,208
1995	6,648	2,330	75	14	2	9,069
1996	6,182	1,843	105	16	5	8,151
avg 1992–1996	6,542	2,284	110	18	4	8,958

Source: Adapted from API 1998b

Table 5-2. Total Number of Oil Spill from Vessels: 1984–1996

Year	Under 10 Gallons	10–999 Gallons	1,000–9,999 Gallons	10,000–99,999 Gallons	More than 100,000 Gallons	Total
1984	1,333	1,079	78	21	13	2,524
1985	1,178	952	59	13	7	2,209
1986	1,661	1,260	74	15	8	3,018
1987	1,723	1,228	62	13	7	3,033
1988	1,896	1,299	57	23	7	3,282
1989	2,197	1,440	66	25	5	3,733
1990	2,576	1,533	92	28	7	4,236
1991	2,689	1,446	70	12	2	4,219
1992	3,910	1,687	49	6	1	5,653
1993	4,098	1,520	67	8	2	5,695
1994	3,759	1,545	68	11	2	5,385
1995	3 932	1,530	44	11	2	5,519
1996	3,722	1,201	60	10	3	4,996
avg 1992–1996	3,884	1,497	58	9	2	5,450

Source: Adapted from API 1998b

Table 5-3. Total Number of Oil Spill from Facilities: 1984–1996

Year	Under 10 Gallons	10–999 Gallons	1,000–9,999 Gallons	10,000–99,999 Gallons	More than 100,000 Gallons	Total
1984	4,113	2,194	288	53	3	6,651
1985	3,032	1,619	252	39	2	4,944
1986	2,076	1,025	54	10	3	3,168
1987	1,821	1,022	66	10	1	2,920
1988	1,730	939	68	6	4	2,747
1989	2,827	1,140	70	12	2	4,051
1990	3,904	1,187	72	15	4	5,182
1991	4,102	1,174	68	13	1	5,358
1992	2,412	869	56	16	2	3,355
1993	2,799	796	53	8	1	3,657
1994	2,900	831	76	10	6	3,823
1995	2,716	800	31	3	0	3,550
1996	2,460	642	45	6	2	3,155
avg 1992-1996	2,657	788	52	9	2	3,508

Source: Adapted from API 1998b

Table 5-4. Nature of Oil Spill by Material or Product: 1984–1996[a] (Volume in Thousands of Gallons)

Material	Number of spills 1984–1993[a] *(avg annual)*	Number of Spills 1996	Volume of spills 1984–1993[a] *(avg annual)*	Volume of spills 1996
Residuals	3,478 *(348)*	132	6,135 *(614)*	276
Distillates	27,035 *(2,704)*	2,403	12,776 *(1,277)*	2,256
Crude oils	15,973 *(1,597)*	1,450	30,364 *(3,036)*	195
Lubricants	7,563 *(756)*	634	1,784 *(178)*	24
Light ends	104 *(10)*	–	124 *(12)*	–
Miscellaneous	11,925 *(1,193)*	–	2,308 *(231)*	–
Unknown	4,728 *(473)*	–	239 *(24)*	–
Gasoline	5,858 *(586)*	454	3,583 *(358)*	291
Asphalt	489 *(49)*	24	414 *(41)*	20
Jet fuel	1,011 *(101)*	65	1,111 *(111)*	18
Bilge oil	311 *(31)*	177	6 *(0.6)*	2
Benzene and related aromatics	88 *(9)*	11	148 *(15)*	8
Naphthas	338 *(34)*	10	268 *(27)*	3
Kerosene	478 *(48)*	18	542 *(54)*	1
Liquified natural gas	29 *(3)*	–	0	–
Waxes	18 *(2)*	–	4 *(0.4)*	–
Liquified petroleum gas	37 *(4)*	–	15 *(1.5)*	–
All other		2,773		114
Total	79,463 *(7,946)*	8,151	59,820 *(5,982)*	3,208

[a] Data not available for 1994 and 1995

Source: Adapted from API 1998b

119,000 confirmed instances of underground releases of gasoline or similar petroleum bulk fuels to soils or groundwater, with the total number of sites needing remediation likely to climb to over 176,000 by the turn of the century (EPA 1994a). While tests to confirm contamination may involve TPH or tests for surrogates of specific chemicals such as benzene, the UST program does not attempt to make detailed estimates of releases to environmental media.

Since many releases of petroleum to environmental media involve unintentional leakage or spillage, it can be helpful to present some rough estimates of release from various categories of activities or components within the overall petroleum production and distribution system. Results assembled from various sources in a study by Doyle (1994) are summarized in Table 5-5. While different estimation techniques could alter these leakage values, the major components of the oil production and distribution system include: leaking (abandoned) oil wells, large aboveground storage tanks, leaks from gasoline stations, tank bottoms and refinery residuals disposal, used motor oil, and evaporative losses.

Doyle (1994) estimates the total amount of leakage or spillage related to petroleum product production, processing, and distribution to end users at around 134 million barrels per year (see Table 5-5); different estimation approaches could lead to slightly different total figures. For instance, total U.S. refinery output in 1993 was around 2,608 million gallons (PennWell 1994); total consumption of motor gasoline during 1992 was around 5,762 million barrels (PennWell 1994). The levels of spillage or leakage resulting in releases to the environment amount to about 2.3% of total refinery output and around 5% of total gasoline consumed; conversely, about 95% of the original amounts petroleum products are completely consumed, generally in combustion processes to heat homes or power cars, trucks, planes, boats, and trains.

Recurrent spills or a long history of disposal at specific sites can lead to concerns. Oil dumped onto soils can saturate the soil matrix (see Section 5.3). This type of very concentrated contamination can be virtually impossible to eliminate without excavating and removing all the soil materials.

If TPH is introduced at any depth within the soil matrix, as in the case of leaks from underground storage tanks, natural weather and biodegradation processes are rendered less effective and the chances are increased that some of the TPH fractions may contaminate groundwater. Since many

Table 5-5. Estimated Releases From Components of the Oil System (Annual Estimates in Millions of Barrels per Year)

Type of release	Size of release	Major media impacted	Description of category
Oilfield spills	1.050 (<1%)	Soil Surface water Groundwater	Producing wells and tank batteries.
Leaking oil wells	3.650 (2.7%)	Soil Surface water Groundwater	Older "abandoned" wells never capped; up to 1.2 million such wells in the United States.
Oil in waste pits or produced water	1.200 (<1%)	Soil Groundwater	Buried or land applied wastes from producing wells or exploration activities.
Aboveground tanks	63.875 (47.4%)	Soil, Air, Groundwater	Usually larger tank batteries, often part of interstate pipeline systems.
Existing underground plumes	1.200 (<1%)	Groundwater Soil	Tank farms, transhipment terminals, and refineries with large amounts of "free product" beneath the facilities. At least 356 facilities currently pump from the largest plumes.
Pipelines	0.714 (<1%)	Surface water Soil	Larger interstate pipelines and low pressure gathering systems from smaller tank batteries.
Leaks from gas stations	5.200 (3.9%)	Soil Groundwater	At least 25% of the nation's filling stations may face remediation under the UST program.
Tank bottoms & refinery waste	24.200 (17.9%)	Soil	Heavier residuals and sludges from refineries.
Used motor oil	14.000 (10.4%)	Water Soil	The U.S. generates about 1.4 billion gallons of used motor oil per year. Less than half is re-refined. Much "home fix-it" oil is not disposed of properly.
Oil spills in U.S. waters	1.095 (<1%)	Surface water	Tankers, barge, and pipeline accidents, mostly during vessel loading or unloading operations.
Oil & grease discharge	0.090 (<1%)	Surface water	Mostly from offshore drilling in near coastal waters.
Operational discharges from tankers	0.178 (<1%)	Surface water	Discharge of cargo and bilge oil in near coastal waters.
Evaporative losses	18.428 (13.7%)	Air	Transfers at refineries or tankers, losses at storage facilities, and during vehicle fueling. Up to 18 grams of hydrocarbons vented to air for each gallon of gasoline used.
Total	134.880		

Source: Adapted from Doyle 1994

TPH components have densities less than or close to that of water, these lighter nonaqueous phase liquids (LNAPLs) generally pose less potential for groundwater pollution than most chlorinated solvents (e.g., PCBs or TCE) that are denser than water (denser nonaqueous phase liquids [DNAPLs]). The nonaqueous phase liquid refers to liquids that are immiscible in water. Still, there are risks for shallow groundwater supplies, which may be used for private wells for drinking water purposes.

For surface water, the relatively low density of many petroleum fractions can pose some major short-term concerns, especially for fish and wildlife. Many petroleum fractions float in water and form thin surface films (Jordan and Payne 1980; Mackay 1984). Gasoline, diesel, or other common fuel oils when spilled to water quickly spread out into a film 0.1 millimeter or less in thickness. This means that a very small amount of oil can create a film over a very large area of water surface. While natural physical and biological weathering processes will dissipate or degrade such oil slicks in time frames ranging from days to a few weeks, there is considerable short-term opportunity for damage to water fowl, aquatic mammals, fish, and other aquatic organisms. For inland waters, large oil spills may force shutdowns in surface water withdrawals for public drinking water supplies until the surface slicks have dissipated (Clark et al. 1990). Where the spilled petroleum washes up onto beaches or shorelines, there may be short-term damage to fish and wildlife as well as impacts to recreational use of shoreline or riparian areas for human swimming or fishing.

Some heavier petroleum fractions, including the chemicals called PAHs found in motor oils or as byproducts of combustion, show neutral buoyancy or may be heavier than water. Such components can accumulate in substrates. This can lead to stresses for benthic organisms, shellfish, or bottom feeding fish. PAHs or “tarballs” formed when lighter oil fractions combine with suspended sediment or algae can have a serious impact on a water body’s use for commercial fishing or shellfishing and its value for recreational swimming or sports fishing.

In addition to releases from the various components or activities that make up the production and distribution system for petroleum products (the oil system), many older waste sites show TPH-related site contamination. Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) site descriptions often mention petroleum, oil and grease, or petroleum, oil, and lubricants (POL) as present at a former waste disposal site. An example is given below for a waste oil recycling site, where TPH-type chemicals were obviously a common site contaminant. The CERCLA clean-up actions, however, focus on a range of specific hazardous or toxic chemicals. Some of the specific chemicals (e.g., toluene) would show up in a TPH test, but the chlorinated solvents and metals do not.

Since a site cannot be prioritized for CERCLA attention if the only problem involves TPH site contamination, CERCLA actions are often triggered by the presence of other site contaminants that can clearly be ranked as hazardous or toxic. The long-term clean-up actions may entail remediation steps that reduce or eliminate the TPH concerns, but these actions are secondary results of the cleanup activities.

This oil exemption aspect of CERCLA introduces complications when trying to present summary data on the distribution of NPL sites showing TPH site contamination. The ATSDR HazDat database contains only 34 records dealing explicitly with TPH at current or former NPL sites (ATSDR 1998a).

While CERCLA deals with former waste disposal sites, the RCRA program handles active waste disposal facilities. Many waste facilities still in use have older sites (management units) within them that may need corrective actions similar to those encountered at CERCLA NPL sites. At least 5,100 RCRA hazardous waste treatment, storage, and disposal facilities (TSDFs) may need some corrective actions before they can be shut down (EPA 1994b). While there are no readily available statistics, many of these RCRA facilities needing corrective actions also contain waste oils (EPA 1994b). As with CERCLA, RCRA works with what amounts to a hazardous waste exclusion clause for ordinary petroleum products. Most clean-up efforts, therefore, focus on legally defined toxics and hazardous materials as the main line of attack in site remediation, with the expectation that these measures will also help ameliorate any TPH-related concerns.

5.3 FATE AND TRANSPORT

5.3.1 Overview

Chemical analysis for all individual compounds in a petroleum bulk product released to the environment is generally unrealistic due to the complexity of these mixtures and the laboratory expense. Determining the chemical composition of a petroleum release is further complicated by hydrodynamic, abiotic, and biotic processes that act on the release to change the chemical character. The longer the release is exposed to the environment, the greater the change in chemical character and the harder it is to obtain accurate analytical results reflecting the identity of the release. After extensive weathering, detailed knowledge of the original bulk product is often less valuable than current site-specific information on a more focused set of hydrocarbon components, for example TPH fractions.

Health assessment efforts are frequently frustrated by three primary problems: (1) the inability to identify and quantify the individual compounds released to the environment as a consequence of a petroleum spill; (2) the lack of information characterizing the fate of the individual compounds in petroleum mixtures; and (3) the lack of specific health guidance values for the majority of chemicals present in petroleum products. To define the public health implications associated with exposure to petroleum hydrocarbons, it is necessary to have a basic understanding of petroleum properties, compositions, and the physical, chemical, biological, and toxicological properties of the compounds most often identified as the key chemicals of concern.

5.3.2 Fate and Transport Processes

This section describes important chemical, physical, and biological processes that affect the behavior of hydrocarbon compounds in the environment. This information may be used to identify the environmental media that are likely to be affected by a release and to predict the potential for subsequent human exposure.

5.3.2.1 Bulk Oil Migration

Petroleum products released to the environment migrate through soil via two general pathways: (1) as bulk oil flow infiltrating the soil under the forces of gravity and capillary action, and (2) as individual compounds separating from the bulk petroleum mixture and dissolving in air or water. When bulk oil flow occurs, it results in little or no separation of the individual compounds from the product mixture and the infiltration rate is usually fast relative to the dissolution rate (Eastcott et al. 1989). Many compounds that are insoluble and immobile in water are soluble in bulk oil and will migrate along with the bulk oil flow. Factors affecting the rate of bulk oil infiltration include soil moisture content, vegetation, terrain, climate, rate of release (e.g., catastrophic versus slow leakage), soil particle size (e.g., sand versus clay), and oil viscosity (e.g., gasoline versus motor oil).

As bulk oil migrates through the soil column, a small amount of the product mass is retained by soil particles. The bulk product retained by the soil particles is known as “residual saturation.” Depending upon the persistence of the bulk oil, residual saturation can potentially reside in the soil for years (Dragun 1988). Residual saturation is important as it determines the degree of soil contamination and can act as a continuing source of contamination for individual compounds to separate from the bulk product and migrate independently in air or groundwater (Bauman 1988). If

the release is persistent in the environment, there can be impacts to extensive areas as the individual compounds continue to separate and migrate away from the spill area via air or groundwater.

When the amount of product released to the environment is small relative to the volume of available soil, all of the product is converted to residual saturation and downward migration of the bulk product usually ceases prior to affecting groundwater resources. Adverse impacts to groundwater may still occur if rain water infiltrates through soil containing residual saturation and initiates the downward migration of individual compounds.

When the amount of product released is large relative to the volume of available soil, the downward migration of bulk product ceases as water-saturated pore spaces are encountered. If the density of the bulk product is less than that of water, the product tends to "float" along the interface between the water saturated and unsaturated zones and spread horizontally in a pancake-like layer, usually in the direction of groundwater flow. Almost all motor and heating oils are less dense than water (Knox 1993; Mackay 1988) and are referred to as LNAPLs.

If the density of the bulk product is greater than that of water, the product will continue to migrate downward through the water table aquifer under the continued influence of gravity. Downward migration ceases when the product is converted to residual saturation or when an impermeable surface is encountered. Polychlorinated biphenyls and other chlorinated organic solvents are usually denser than water and are characterized as DNAPLs.

In reality, bulk oil flow is affected by numerous product-specific and site-specific factors. As a consequence, product distribution in the subsurface can be quite complex.

5.3.2.2 Compound Migration

As the bulk product migrates through the soil column, individual compounds may separate from the mixture and migrate independently. Chemical transport properties such as volatility, solubility, and sorption potential are often used to evaluate and predict which compounds will likely separate from the mixture.

Volatility. Volatility is defined as the propensity of a chemical to partition to air and migrate as a vapor. It is primarily a function of the vapor pressure of the compound. Vapor pressure is defined as the pressure of a chemical exerted by its vapor when in equilibrium with the solid or liquid form of

that chemical. For example, if a chemical in a liquid form is placed in a closed container, molecules of the chemical that possess relatively high kinetic energy will migrate to the surface of the liquid and evaporate into the air space in the container.

Since petroleum products are complex mixtures of hundreds of compounds, the compounds characterized by relatively high vapor pressures tend to volatilize and enter the vapor phase. The exact composition of these vapors depends on the composition of the original product. Using gasoline as an example, compounds such as butane, propane, benzene, toluene, ethylbenzene and xylene are preferentially volatilized (Bauman 1988). Because volatility represents transfer of the compound from the product or liquid phase to the air phase, it is expected that the concentration of that compound in the product or liquid phase will decrease as the concentration in the air phase increases.

In general, compounds having a vapor pressure in excess of 10^{-2} mm Hg are more likely to be present in the air phase than in the liquid phase. Compounds characterized by vapor pressures less than 10^{-7} mm Hg are more likely to be associated with the liquid phase. Compounds possessing vapor pressures that are less than 10^{-2} mm Hg, but greater than 10^{-7} mm Hg, will have a tendency to exist in both the air and the liquid phases (Knox 1993).

Although volatility is a function of vapor pressure, environmental factors affect the rate of volatilization. For example, high summer temperatures enhance volatilization, particularly when soils begin to dry out. The rate of volatilization is also a function of air and soil temperature, humidity, wind speed, soil type, moisture content, oil composition, solar radiation, and thickness of the oil layer. Volatilization of benzene, toluene, ethylbenzene, and xylene from gasoline-contaminated soils tends to increase with decreasing moisture content (Frankenberger 1992). Bossert and Bartha (1986) indicated that *n*-alkanes greater than C_{18} exhibit no substantial volatilization at ambient temperatures; however, lighter fractions ($<C_{18}$) are subject to volatilization.

The propensity for a compound to volatilize from an aqueous phase can be grossly estimated using Henry's law, which relates vapor pressure, solubility, and molecular weight. Henry's law constant can be estimated using these three chemical-specific parameters or it can be measured on a compound-by-compound basis in the laboratory. Henry's law constant is frequently used to assess the environmental fate of organic compounds in the subsurface.

Solubility. Solubility is one of the key factors in determining compound behavior, and thus the impact, of a chemical in the environment. Solubility is expressed in terms of the number of milli-

grams of pure chemical that can be dissolved in one liter of water under standard conditions of 25 ºC and one atmosphere of pressure. The solubility of an organic compound determines its propensity to dissolve into water. The greater the solubility, the greater the likelihood that the chemical will dissolve into infiltrating rainwater or groundwater and migrate away from the release area.

Solubility generally decreases with increasing molecular weight of the hydrocarbon compounds. For compounds having similar molecular weights, the aromatic hydrocarbons are more water soluble and mobile in water than the aliphatic hydrocarbons (ASTM 1995) and branched aliphatics are less water-soluble than straight-chained aliphatics. Coleman et al. (1984) determined that the compounds most likely to be measured in water in contact with gasoline, kerosene, and fuel oil #2 were the light-fraction, aromatic hydrocarbons such as benzene, toluene, ethylbenzene, and xylenes. They found that although the aromatic compounds in these three fuels may comprise as much as 50% by weight, aromatic compounds in the C_6-C_{13}, range made up approximately 95% of the compounds dissolved in water. This correlates well with studies showing an enrichment of light-fraction hydrocarbons in the water phase and a depletion in the fuel phase.

It is important to note that the partitioning behavior of organic compounds is affected by the presence of other hydrocarbons in the subsurface. The maximum dissolved concentrations achieved in the subsurface are always less than the concentration of any pure compound, when it is present as one of many constituents of a petroleum product (ASTM 1995). For example, the solubility of pure benzene in water is given as 1,780 mg/L, but the maximum calculated concentration in an aquifer immediately beneath a gasoline release has been estimated to be about 62 mg/L (Daugherty 1991).

Organic Carbon-Water Partition Coefficient. The organic carbon-water partition coefficient (K_{oc}) describes the propensity for a organic compound to partition between water and organic carbon in the soil. Chemical mobility can be determined based on the likelihood of a chemical to partition more strongly to either the organic carbon in the substrate or the water. If the chemical is strongly associated with the substrate (i.e., sorbed), the chemical is relatively immobile and will not be leached or transported great distances from the area of the release. In contrast, if the chemical is weakly sorbed to the substrate, the chemical has the potential to be transported greater distances and greater chance to contact human receptors.

The degree of sorption not only affects the mobility of the compound, it can also affect other transport and transformation reactions. For example, volatilization and biodegradation rates are directly dependent upon the extent of partitioning (Dragun 1988). A compound that is strongly

sorbed to the organic carbon in the substrate is less available and less likely to be volatilized or biodegraded.

A mobility classification scheme based on the K_{oc} indicates that compounds having K_{oc} values <50 L/kg, 50-150 L/kg, and 150-500 L/kg are considered to be very mobile, mobile, and intermediate in mobility, respectively (Dragun 1988). Using this scheme, benzene (K_{oc} = 60 L/kg) is classified as mobile; whereas toluene, ethylbenzene, and total xylenes (K_{oc} = 182 L/kg, K_{oc} = 363 L/kg, and $K_{oc} \cong$ 400 L/kg, respectively) are classified as having intermediate immobility.

In summary, lighter petroleum products such as gasoline contain constituents with higher water solubility and volatility and lower sorption potential than heavier petroleum products such as fuel oil. Data compiled from gasoline spills and laboratory studies indicate that these light-fraction hydrocarbons tend to migrate readily through soil, potentially threatening or affecting groundwater supplies. In contrast, petroleum products with heavier molecular weight constituents, such as fuel oil, are generally more persistent in soils, due to their relatively low water solubility and volatility and high sorption capacity (Stelljes and Watkin 1991).

5.3.2.3 Biodegradation

Indigenous microbes found in many natural settings (e.g., soils, groundwater, ponds) have been shown to be capable of degrading organic compounds. Biodegradation occurs as microbes use organic compounds as a source of energy. Unlike other fate processes that disperse contaminants in the environment, biodegradation can eliminate the contaminants without transferring them across media. The final products of microbial degradation are carbon dioxide, water, and microbial biomass.

The rate of hydrocarbon degradation depends on the chemical composition of the product released to the environment as well as site-specific environmental factors. Generally the straight chain hydrocarbons and the aromatics are degraded more readily than the highly branched aliphatic compounds (Havlicek 1988). The *n*-alkanes, *n*-alkyl aromatics, and the aromatics in the C_{10}-C_{22} range are the most readily biodegradable; *n*-alkanes, *n*-alkyl aromatics, and aromatics in the C_5-C_9 range are biodegradable at low concentrations by some microorganisms, but are generally preferentially removed by volatilization and thus are unavailable in most environments; *n*-alkanes in the C_1-C_4 ranges are biodegradable only by a narrow range of specialized hydrocarbon degraders; and *n*-alkanes, *n*-alkyl aromatics, and aromatics above C_{22} are generally not available to degrading microorganisms. Hydrocarbons with condensed ring structures, such as PAHs with four or more

rings, have been shown to be relatively resistant to biodegradation. PAHs with only 2 or 3 rings (e.g., naphthalene, anthracene) are more easily biodegraded (Park et al. 1990).

A large proportion of the water-soluble fraction of the petroleum product may be degraded as the compounds go into solution. As a result, the remaining product may become enriched in the alicyclics, the highly branched aliphatics, and PAHs with many fused rings.

Environmental factors such as oxygen content, pH, moisture content, temperature, nutrient concentrations, and the microbiota also affect the rate of biodegradation. In almost all cases, the presence of oxygen is essential for effective biodegradation of oil. Anaerobic decomposition of petroleum hydrocarbons leads to extremely low rates of degradation (Frankenberger 1992). The ideal pH range to promote biodegradation is close to neutral (6-8). For most species, the optimal pH is slightly alkaline, that is, greater than 7 (Dragun 1988). The moisture content of the contaminated soil will affect biodegradation of oils due to dissolution of the residual compounds, dispersive actions, and the need for microbial metabolism to sustain high activity. The moisture content in soil affects microbial locomotion, solute diffusion, substrate supply, and the removal of metabolic by-products. Excessive moisture will limit the gaseous supply of oxygen for enhanced decomposition of petroleum hydrocarbons. Most studies indicate that optimum moisture content is within 50-70% of the water holding capacity (Frankenberger 1992).

All biological transformations are affected by temperature. Generally, as the temperature increases, biological activity tends to increase up to a temperature where enzyme denaturation occurs. The presence of oil should increase soil temperature, particularly at the surface. The darker color increases the heat capacity by adsorbing more radiation. The optimal temperature for biodegradation to occur ranges from 18 ºC to 30 ºC. Minimum rates would be expected at 5 ºC or lower (Frankenberger 1992).

There are at least 11 essential macronutrient and micronutrient elements that must be present in the soil in proper amounts, forms, and ratios to sustain microbe growth (Dragun 1988). These 11 elements are nitrogen, phosphorus, potassium, sodium, sulfur, calcium, magnesium, iron, manganese, zinc, and copper. Nitrogen is usually the main limiting nutrient governing the rate of decomposition of petroleum hydrocarbons. However, small amounts of phosphorus fertilizers may also be necessary to stimulate biodegradation (Mills and Frankenberger 1994).

Biodegradation rates in soils are also affected by the volume of product released to the environment. At concentrations of l-0.5% of oil by volume, the degradation rate in soil is fairly independent of oil concentrations. However, as oil concentration rises, the first order degradation rate decreases and the oil degradation half-life increases. Ultimately, when the oil reaches saturation conditions in the soil (i.e., 30-50% oil), biodegradation virtually ceases (Eastcott et al. 1989). This is substantiated by Borden et al. (1986) who found that biodegradation of trace quantities of hydrocarbon compounds occurred along contaminant plume edges in the presence of oxygenated formation water, but that little biodegradation occurred in the plume center where concentrations were higher. Wilson et al. (1985) also found biodegradation to take place selectively along plume margins controlled by the oxygen supply.

The point at which biodegradation starts to become adversely affected by the amount of oil present is not well established. Other inhibitory effects include the generation of toxic intermediate organic compounds. Degradation of aromatic hydrocarbons, such as toluene, can yield phenolic and benzoic acid intermediates. Various microbial populations may be inhibited by compounds such as phenol and toluene, particularly at high concentrations. Although phenol- and toluene-degrading microorganisms have been isolated in soil exposed to low concentrations of these compounds, they are biocidal at elevated concentrations (Frankenberger 1992).

The inhibitory effects of heavy metals can also influence biodegradation of organic materials. The presence of heavy metals in oil sludge, motor oil, and used crankcase oil may have deleterious effects on the hydrocarbon oxidizers in decomposing petroleum hydrocarbons (Frankenberger 1992). Jensen (1977) studied the effects of lead on biodegradation of oily waste in soil and found that the presence of lead caused certain changes in the population of soil microbiota. Reduction in the bacterial population was evident, particularly at the highest lead concentration of 5,000 ppm. Measurements of oxygen consumption revealed increased microbial activity after the addition of oil to soils, but the presence of lead markedly reduced this activity with a prolonged lag phase in the biodegradation of oil sludge. Other elements of concern include zinc, copper, chromium, nickel, and cadmium. With repeated applications of oily sludge to a landfarm operation, heavy metals may accumulate at levels in which biodegradation may be reduced.

5.3.3 Models

An understanding of the factors that affect the fate and transport of contaminants in the environment, and the ability to develop and apply mathematical models that incorporate these factors, are important in risk management applications. Models are used to approximate real world processes to provide environmental analyses to support management decisions. If properly used, models can assist decision makers in effectively dealing with the complex issues related to petroleum releases in the environment.

As noted earlier, petroleum products released to the environment migrate through soil by two general pathways: via bulk oil flow and as individual compounds dissolved in air or water. Although comprehensive mathematical models could be devised to treat both types of migration, the resultant framework would likely be excessively complex. Eastcott et al. (1989) suggests a two-stage modeling approach. The first stage considers transport of the bulk oil phase. After the oil is rendered immobile, a second stage is applied to assess the fate of the individual compounds that separate from the bulk phase. Models of this sort are often called solute transport models. The use of a two-stage approach is justified because when bulk oil flow does occur, it results in little or no component separation (i.e., benzene travels as fast as hexane) and the transport rate is usually fast relative to that of the dissolution rate (Eastcott et al. 1989).

Modeling the bulk oil phase is complex and includes many uncertainties; consequently, it has not been employed extensively in decision-making processes (Bonazountas 1988). One exception is the Hydrocarbon Spill Screening Model (HSSM), which simulates the flow of LNAPLs through the unsaturated zone, LNAPL spreading in the capillary fringe, and the transport of a single chemical originating from the LNAPL in the water table aquifer (Charbeneau et al. 1995). HSSM may be used to provide an estimate of dissolved concentrations of compounds originating from a petroleum release of known composition, rate of release, and volume of release. It is generally assumed that modeling interest lies in the potential for adverse impacts to water quality, and most modeling practices have concentrated on the behavior of dissolved organic compounds at the edges of bulk oil plumes or lenses. Table 5-6 lists selected soil and groundwater models that are well documented, operational, and representative of types of available models.

Table 5-6. Selected Soil and Groundwater Models

Model	Model category	Description
PRZM-2	Unsaturated zone/groundwater	PRZM-2 is a combination of two models developed to simulate the one-dimensional movement of pesticides. It has been used predominantly for evaluation of pesticides in the root zone.
SESOIL	Unsaturated zone	SESOIL is a one-dimensional, finite difference flow and transport model. The model estimates the rate of vertical solute transport and transformation from the land surface to the water table.
VLEACH	Unsaturated zone	VLEACH is a one-dimensional, finite difference model developed to simulate the transport of contaminants displaying linear partitioning behavior through the vadose zone to the water table by aqeuous advection and diffusion.
MULTIMED	Unsaturated zone/groundwater	MULTIMED was developed as a multimedia fate and transport model to simulate contaminant migration from waste disposal units. Release to either air or soil, including the unsaturated and the saturated zones, are possible interception of the subsurface contaminant plume by a surface stream are included.
M3TD	Groundwater	MT3D is a transport model that simulates advection, dispersion, source/sink mixing, and chemical reactions of contaminants in groundwater flow systems in either two or three dimensions.
MODFLOW	Groundwater	MODFLOW is a three-dimensional finite-difference groundwater flow model. This model analyzes groundwater flow under various hydrologic conditions, including a combination of hydrgeologic layers and external stresses.
PATH3D	Groundwater	PATH3D is a general particle tracking program for calculating groundwater paths and travel times in steady-state or transient, two- or three-dimensional flow fields. This program is particularly useful for delineating contaminant capture zones or wellhead protection zones.
CHEMFLO	Unsaturated zone	CHEMFLO is a one-dimensional flow and transport model designed to simulate the movement of water and chemicals into and through soils.
MINTEQ	Geochemical	MINTEQ is a geochemical model designed to estimate equilibrium compositions of dilute aqueous solutions.
HSSM	Unsaturated zone/groundwater	HSSM simulates the flow of light nonaqueous phsase liquids (LNAPLs) through the unsaturated zone, spreading in the capillary fringe, and transport of chemical constituents originating fron the LNAPLs in the water table aquifer.
SCDM	Ranking	

Source: Bonazountis 1988, 1991; Charbeneau 1995; Daugherty 1991

5.3.3.1 Transport Equations

Using the fraction method, hydrocarbon fractions are established on the basis of compounds having similar leaching and volatilization factors. The TPHCWG (1997b) used equations from the *Standard Guide for Risk-Based Corrective Action (RBCA) Applied to Petroleum Release Sites* (ASTM 1995) to estimate the volatilization factor and leaching factor. These equations are shown below.

$$\text{Volatilization Factor } \frac{[\frac{mg}{m^3} - \text{air}]}{[\frac{mg}{kg} - \text{soil}]} = \frac{H\rho_s}{[\theta_{ws}+K_s\rho_s+H\theta_{as}]\ [1+\frac{U_{air}\delta_{air}L_s}{D_{eff}W}]} \times 10^3 \qquad [5\text{-}1]$$

$$\text{Leaching Factor } \frac{(\frac{mg}{L}-H_2O)}{(\frac{mg}{kg})-\text{soil})} = \frac{\rho_s}{[\theta_{ws}+K_s\rho_s+H\theta_{as}][1+\frac{U_{gw}\delta_{gw}}{IW}]} \times 10^0 \qquad [5\text{-}2]$$

where

ρ_s = soil bulk density (g/cm^3)
θ_{as} = volumetric air content in vadose zone soils (cm^3/cm^3)
θ_{ws} = volumetric water content in vadose zone soils (cm^3/cm^3)
U_{air} = wind speed above ground surface (c/s)
δ_{air} = ambient air mixing zone thickness (cm)
L_s = depth to subsurface soil sources (cm)
W = width of source area parallel to groundwater flow direction (cm)
U_{gw} = groundwater Darcy velocity (cm/yr)
δ_{gw} = groundwater mixing zone thickness (cm)
I = infiltration rate of water through soil (cm/yr)
K_s = soil-water sorption coefficient (f_{oc} K_{oc}) (cm^3/g)
H = Henry's law constant (cm^3/cm^3)
D_{eff} = effective diffusion coefficient through soil (cm^2/s)

In accordance with the RBCA conceptual models, the leaching factor and the volatilization factor were estimated for 260 compounds present in petroleum distillates and crude oil.

5.3.3.2 Estimating Physical and Chemical Properties

Solution of equations 5-1 and 5-2 requires site-specific values as well as chemical-specific values. Critical chemical-specific properties in the above equations are the Henry's law constant (H), the organic carbon/water partition coefficient (K_{oc}), and the effective diffusion coefficient through soil (D_{eff}). Henry's law constants are estimated using solubility and vapor pressure values obtained from data compilations. The K_{oc} is estimated from the octanol-water coefficient (K_{ow}) and is commonly estimated from the Hansch and Leo (1979) fragment constant approach (TPHCWG 1997b):

Diffusivity in air (D_{eff}) for compounds where experimental data were unavailable can be calculated using the following equation:

$$D_{air\text{-}B} = \frac{J_B}{\nabla X_B} \qquad [5\text{-}3]$$

where

$D_{air\text{-}B}$ = diffusivity of compound B in air (cm^2/s)
J_B = net molal flux of B ($mol/cm^2\text{-}s$)
X_B = concentration gradient of B ($mol/\ cm^3\text{-}cm$)

For a more detailed descriptions of K_{oc} and diffusivity refer to TPHCWG (1997b).

The TPHCWG correlated chemical-specific leaching factors and volatilization factors for hydrocarbon compounds present in gasoline and crude oil with the Carbon Number Index, which is defined as the boiling point normalized to the *n*-alkanes. The correlation to Carbon Number Index was used because it closely follows chemical behavior in the boiling point gas chromatograph column and because the literature indicates that the various properties should be well correlated to this index (Gustafson 1995).

The leaching and volatilization behavior of the hydrocarbon compounds spans many orders of magnitude. Compounds exhibiting similar leaching and volatilization factors were grouped into carbon fractions by the TPHCWG. Compounds were assigned to a given fraction within aliphatic and aromatic groupings on the basis of having leaching and volatilization factors within

approximately one order of magnitude (TPHCWG 1997b). A set of 13 fractions were selected by the TPHCWG for use in evaluating TPH environmental levels (TPHCWG 1997b).

5.3.3.3 Transport Models

Transport models fall into four main categories:

- unsaturated zone,
- groundwater,
- geochemical, and
- ranking

The first two categories use similar methodologies applied to different geohydrologic conditions; the third estimates chemical concentrations at equilibrium; and the fourth serves as a screening methodology to evaluate the severity of a release. As shown in Table 5-6, the majority of the models are geared toward assessing contaminant behavior in the unsaturated zone and groundwater. These types of models can appropriately be used for assessing petroleum release sites, but it is important to note that they have been developed for the broad spectrum of contaminants typically found at Superfund sites (e.g., chlorinated hydrocarbons and metals) and have not always been verified or validated for petroleum hydrocarbons in natural porous media (Daugherty 199 1). For example, well known and available models such as SESOIL, which is a one-dimensional, finite difference flow and transport model developed for evaluating the migration of contaminants through the unsaturated zone, cannot simulate the transfer of hydrocarbon compounds from the bulk oil phase to the dissolved aqueous phase.

Despite their limitations, models are useful for assessing generic effects of contaminants or impacts over larger areas. For this purpose, however, simplified expressions derived from first principles appear to be as useful as more elegant computer models for the evaluation of contaminant fate and transport at small sites.

For purposes of this section, the chemical-specific parameters for the petroleum hydrocarbon fractions are based on selecting a midpoint for the fraction, based on empirical data unified by equivalent carbon number (EC). The fractions labeled as C_5-C_7 and >C_7-C_8 are characterized by one compound only, benzene and toluene, respectively. Remaining fractions are characterized by multiple

compounds, as described in TPHCWG (1997b). Representative physical parameters for TPH fractions are presented in Table 5-7.

ASTM's risk-based corrective action (RBCA) uses a tiered approach to data collection and analysis in supporting decisions on site assessment and response to petroleum. The RBCA procedure begins with the assessment of the site (see Figure 5-2 for RBCA process flow-chart).

As part of Tier 1, a look-up table is used to determine whether site conditions satisfy the criteria for a quick regulatory closure or warrant a more site-specific assessment. The look-up table is a tabulation for potential exposure pathways, media (i.e., soil, water, and air), a range of incremental carcinogenic risk levels and hazard quotients equal to unity, and potential exposure scenarios for each chemical of concern. In Tier 2, the non-site-specific assumptions and point(s) of exposure (point at which an individual or population may come in contact with a chemical of concern originating from a site) used in Tier 1 are replaced with site-specific data and information. In Tier 2, the user applies Tier 1 risk-based screening levels (RBSL) look-up table values for the direct exposure scenario at reasonable point(s) of exposure (as opposed to the source areas as is done in Tier 1). The additional site-specific data may support alternate fate and transport analysis. Tier 2 RBCA process also involves the development of site-specific target levels (SSTLs) based on the measured and predicted attenuation of the chemical(s) of concern away from the source using relatively simplistic mathematical models. In Tier 3 evaluation, SSTLs for the source area(s) and the point(s) of compliance are developed on the basis of more sophisticated statistical and contaminant fate and transport analysis using site-specific input parameters for both direct and indirect exposure scenarios. Tier 3 evaluation is much more complex than Tiers 1 and 2 since it may include additional site assessment, probabilistic evaluations, and sophisticated chemical fate/transport models.

5.4 LEVELS IN THE ENVIRONMENT

It is extremely difficult to make general statements about typical TPH or TPH-component levels in environmental media. Environmental fate and transport processes of TPH mixtures are complex. Interactions of the chemicals within the bulk oil typically result in different environmental fate and transport than would be predicted for the individual components. As with the discussion of basic fate and transport processes (see in Section 5.3), site-specific information is nearly always needed for

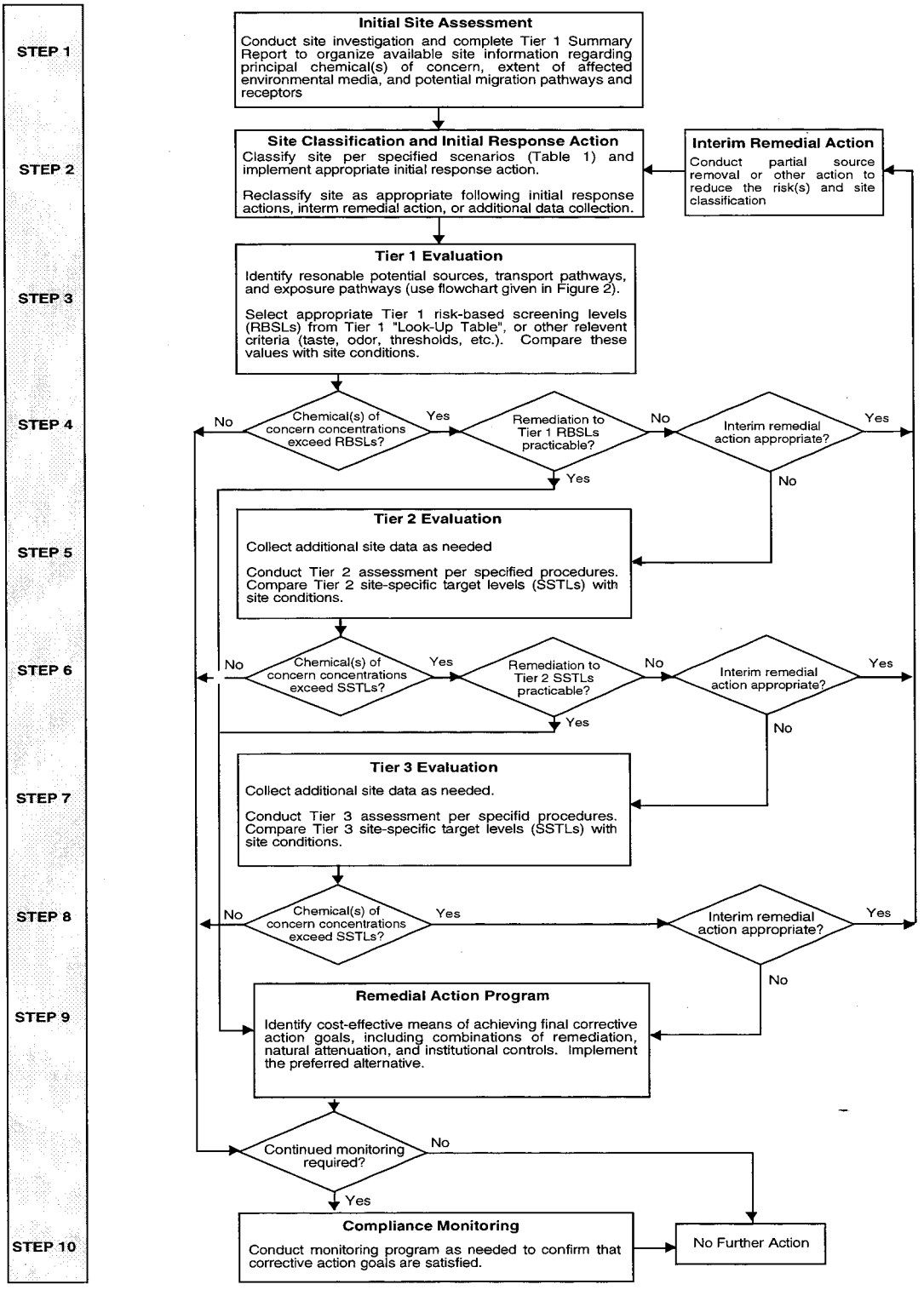
Table 5-7. Representative Physical Parameters for TPH Fractions, Based on Correlation to Relative Boiling Point Index

Fraction	Solubility, mg/L	Vapor pressure, atm	Henry's law, cm^3/cm^3	Log K_{oc}
Aliphatics				
EC_5–EC_6	36	0.35	47	2.9
$EC_{>6}$–EC_8	5.4	0.063	50	3.6
$EC_{>8}$–EC_{10}	0.43	0.0063	55	4.5
$EC_{>10}$–EC_{12}	0.034	0.00063	60	5.4
$EC_{>12}$–EC_{16}	0.00076	0.000076	69	6.7
$EC_{>16}$–EC_{35}	0.0000025	0.0000011	85	8.8
Aromatics				
EC_5–EC_7[a]	220	0.11	1.5	3.0
$EC_{>7}$–EC_8[b]	130	0.035	0.86	3.1
$EC_{>8}$–EC_{10}	65	0.0063	0.39	3.2
$EC_{>10}$–EC_{12}	25	0.00063	0.13	3.4
$EC_{>12}$–EC_{16}	5.8	0.000048	0.028	3.7
$EC_{>16}$–EC_{21}	0.65	0.0000011	0.0025	4.2
$EC_{>21}$–EC_{35}	0.0066	0.00000000044	0.000017	5.1

[a] The only compound contained in this fraction is benzene.
[b] The only compound contained in this fraction is toluene.

Source: TPHCWG 1997b

Figure 5-2. Risk Based Corrective Action Process Flow Chart



Source: ASTM 1995

correct interpretation of data for such media as surface water, soils, or groundwater. Petroleum site contaminants, especially the types of bulk fuel products and lubricants that are the focus of this profile, are usually encountered as liquids or semi-liquid sludges. The site contaminants almost always originate as mixtures of many different hydrocarbons typical of such initial products as motor gasoline, jet fuels, or fuel oils. Frequently, there are portions of a waste site where soils or sub-soil materials have accumulated large masses of petroleum contaminants that form nonaqueous liquid systems. The term nonaqueous phase liquids (NAPL) is often applied to such areas of heavy contamination. NAPLs propagate plumes moving away from the central mass. The NAPL complex, consisting of the central mass and plumes, usually reaches an equilibrium due to a combination of physical, chemical, and biochemical processes. TPH chemicals move into the actual soil or groundwater media from the edge of the NAPL plumes.

Without some knowledge of the locations of NAPL central masses or plumes at a waste site, it can be very hard to interpret analyses from soil samples or test wells. Within the NAPL zone, readings for TPH or one or more specific petrochemicals may be very high. Such high readings are indicative of a soil matrix virtually engulfed by a petroleum waste and represent the bulk oil product rather than the environmental media. Beyond the NAPL zone, the observed levels are substantially less. Media sampling values taken at random from different site-specific spatial contexts should be interpreted within the context of the sampling location relative to the NAPL central masses and plumes.

Since most TPH contamination involves a complex mixture of hydrocarbons, it is unlikely that aqueous readings beyond the NAPL zone will be near the limits of solubility (based on assumptions of a pure hydrocarbon type in equilibrium with water). If concentrations are near or above solubility limits, NAPL was probably present in the sample. TPH materials are relatively insoluble in water, with only the BTEX chemicals or some short-chain aliphatic hydrocarbons showing any appreciable potential for water solubility. When they are part of complex mixtures, the individual components never reach the concentrations predicted from their solubility constants as individual chemicals. For example, chemicals like benzene or toluene, which may constitute a small percentage within an initial bulk product like gasoline, jet fuel, or diesel fuel, have a much greater tendency to stay dissolved in the NAPL system than to become integrated into the water-based system beyond the NAPL boundary. Therefore, the effective solubility of these chemicals as part of a complex mixture is less than it would be in a release of the pure chemical.

Some simple examples help illustrate this point. A study by Burris and MacIntyre (1984) compared the theoretical solubilities of specific chemicals in water to the solubilities of the same chemicals when they were part of such petroleum product mixtures as JP-4 jet fuel. The results are summarized in Table 5-8. Similar comparisons for all the BTEX (benzene, toluene, ethylbenzene, xylene) chemicals, based on materials presented by Potter (1993) are presented in Table 5-9.

The information presented in Tables 5-8 and 5-9 shows that for common petroleum products in water, the solubility of the component chemicals is usually less than the potential solubility of the individual chemical in water by an order of magnitude or more. Table 5-9 also shows why much attention often focuses on site contamination involving gasoline. Gasoline mixtures have much higher percentages of light fraction aromatic hydrocarbons, such as the BTEX aromatics, than other bulk fuel products. This can lead to much higher levels of contamination in ambient water or groundwater from gasoline than from petroleum mixtures with less soluble components. The increased solubilities of the BTEX chemical components from gasoline mixtures would thus be more likely to result in groundwater contamination. For other bulk fuel products, BTEX levels in the mixture are generally far lower; as a result, their water solubility and thus, their potential for groundwater contamination, would be much lower.

In light of these considerations, it becomes easier to see why it is highly desirable that available monitoring data from environmental media be combined with site-specific information. The basic needs are locational data on the spatial configuration of NAPL pockets and plumes combined with analyses of the types of TPH components found in the NAPL system and the surrounding, relatively uncontaminated media (soils, water, and groundwater). With this basic knowledge, a variety of modeling techniques can be applied to estimate effective solubilities of specific hydrocarbon compounds (Feenstra et al. 1991). Moreover, since there can be literally thousands of specific compounds in TPH site contaminants, it is improbable that a site analysis for a TPH-contaminated site would include sampling data for all TPH components. As a result, the surrogate approaches and screening modeling tools such as RBCA have been widely used to evaluate environmental data at TPH-contaminated waste sites.

Table 5-8. Comparison of Select Hydrocarbon Solubilities for JP-4

Compound	Solubility of pure compound in water (mg/L)	Solubility in water when part of JP-4 (mg/L)
Toluene	576.0	28.3
Ethyl benzene	180.0	10.6
n-Octane	0.884	0.173

Source: Burris and MacIntyre 1984

Table 5-9. Benzene, Toluene, Ethybenzene, and Xylenes (BTEX) Concentrations in Water Equilibrated with Various Petroleum Products

Product	Benzene (mg/L)	Toluene (mg/L)	Ethylbenzene (mg/L)	Xylenes (mg/L)
Gasoline	29.5	42.6	2.4	14.7
Diesel fuel	0.13	0.41	0.18	0.70
#6 Fuel oil	0.01	0.03	0.007	0.05
Drinking water standard	0.005 (MCL)	2.0 (MCLG)	0.66 (MCLG)	0.44 (MCLG)
Pure chemical and water	820.0	576.0	180.0	220.0 (160–220)

MCL = maximum contaminant level; MCLG = maximum contaminant level guidelines
Source: Potter 1993

5.5 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of TPH is available. Where adequate information is not available, ATSDR, in conjunction with the NTP, is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of TPH.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

5.5.1 Identification of Data Needs

Physical and Chemical Properties. The physical and chemical properties of some TPH compounds and petroleum products containing TPH are well defined and can be used to estimate the fate of TPH transport fractions following release to the environment (Air Force 1989, 1991; IARC 1987, 1989a, 1989b, 1989c, 1989d). Data needs associated with specific TPH compounds that are components of petroleum products (benzene, ethylbenzene, toluene, xylenes, hexane, and PAHs) are presented in the ATSDR toxicological profiles for these chemicals (ATSDR 1994, 1995d, 1995f, 1997a, 1999a, 1999b).

Production, Import/Export, Use, Release, and Disposal. TPH compounds are the primary component in various petroleum products; therefore, most releases of TPH occur as a result of petroleum product spills either on land or water. More information on the production volumes for various petroleum products, environmental releases, and disposal would aid in assessing the potential for human exposure to TPH as a result of accidental or intentional release. Data needs for specific petroleum products are discussed in the ATSDR toxicological profiles for automotive gasoline (1995a), Stoddard solvent (1995b), jet fuels (1995c and 1998b), fuel oils (1995g), hydraulic fluids (1997b), and mineral-based crankcase oil (1997c).

Environmental Fate. The environmental fate of TPH is based on the environmental partitioning of the major hydrocarbon fractions. However, the environmental fate of chemicals in mixtures and/or bulk oil releases may be different than that observed for releases of individual TPH chemicals (see Sections 5.3.2.1 and 5.3.2.2). The more soluble and volatile fractions (i.e., the low molecular weight aliphatic and aromatic fractions) are more likely to leach to groundwater, volatilize to the air, or biodegrade than the larger TPH compounds are. These higher molecular weight compounds tend to sorb to the soil and persist at the site of release. The movement and persistence of several TPH compounds and petroleum products are well studied. Data needs for specific TPH compounds and petroleum products have been discussed in other ATSDR toxicological profiles (ATSDR 1994, 1995a, 1995b, 1995c, 1995d, 1995e, 1995f, 1995g, 1997a, 1997b, 1997c, 1998b, 1999a, 1999b).

Bioavailability from Environmental Media. The extent of absorption of TPH by inhalation, oral, and/or dermal routes varies because of the wide range of physical/chemical properties observed for these chemicals. The extent of absorption by the various routes depends on the volatility, solubility, lipophilicity, and other properties of the specific TPH chemical or mixture. Several of the TPH component compounds have been discussed in individual ATSDR toxicological profiles (e.g., benzene, ethylbenzene, toluene, xylenes, hexane, PAHs), which should be consulted for further information (ATSDR 1994, 1995d, 1995f, 1997a, 1999a, 1999b). More data linking exposure levels of TPH mixtures with biological levels of component chemicals would be useful in determining which chemicals in the mixture are most likely to be absorbed and by which routes. This information would aid in determining daily human exposure levels and more accurately assessing the risk associated with exposure to TPH.

Food Chain Bioaccumulation. Studies of the accidental and intentional release of gasoline and fuel oils to the aquatic environment indicate that aquatic organisms are able to bioaccumulate some TPH fractions, particularly PAHs (Air Force 1989; Farrington et al. 1982); however, depuration does occur if the source of the contamination is removed (Cox et al. 1975; Williams et al. I989). In general, the lower molecular weight aliphatics and aromatics do not bioaccumulate (Air Force 1989). Further studies are needed to determine the biomagnification potential of the TPH fractions, particularly PAHs, up the food chain within aquatic and terrestrial ecosystems. Specific research needs are presented in the individual ATSDR toxicological profiles on specific hydrocarbon components such as benzene, toluene, total xylenes, and PAHs (ATSDR 1994, 1995d, 1995f, 1997a).

Research on the biomagnification of various petroleum products (e.g., gasoline, fuel oil) would not be useful because the composition of these mixtures changes rapidly in the environment. Individual chemicals present in the original mixture may bioaccumulate, but the mixture does not.

Exposure Levels in Environmental Media. TPH is commonly measured where hydrocarbon releases have occurred (e.g., leaking gasoline, diesel, or fuel oil tanks and petroleum product spills). In most cases, the analytical method does not provide specific information regarding the TPH fractions present (see Section 3.3). More data on levels of TPH fractions and/or their components in the air, water, and soil around facilities where petroleum products are produced, stored, and used would be useful. Data on levels in contaminated surface water, groundwater, and soil are needed to assess the potential risk from these likely sources of exposure.

Exposure Levels in Humans. Workers who use petroleum products in manufacturing and those involved in their transfer may experience increased dermal and inhalation exposures to TPH. Workers in the petroleum refining industry, particularly those involved with monitoring and servicing unit equipment, are known to have increased exposure to TPH (Runion 1988). Reliable monitoring data for levels of TPH in contaminated media could be used in combination with biomarkers to identify TPH exposure and assess the potential risk of adverse health effects in populations living near contaminated areas. This information is necessary for assessing the need to conduct health studies on these populations.

Exposure Registries. No exposure registries for TPH or petroleum products were located. This substance is not currently one of the compounds for which a subregistry has been established in the National Exposure Registry. The substance will be considered in the future when chemical selection is made for subregistries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to exposure to this substance. A registry does exist for benzene, a component of TPH. More information on the benzene exposure registry can be found in the ATSDR toxicological profile for benzene (ATSDR 1997a).

5.5.2 Ongoing Studies

No summary of ongoing studies is presented in this profile. Useful summaries are provided in toxicological profiles for the specific petroleum hydrocarbons or petroleum products, as listed in Table 3-l. As of September 1999, a 90-day toxicity study of a C_9 to C_{16} aromatic fraction in rats and mice was completed by Dr. D. Mattie and colleagues at Wright Patterson toxicology laboratory (DOD), though not published. No other petroleum fraction toxicity research can be reported.

6. HEALTH EFFECTS

6.1 INTRODUCTION

The primary purpose of this chapter is to provide public health officials, physicians, toxicologists, and other interested individuals and groups with an overall perspective on the toxicology of total petroleum hydrocarbons (TPH), and an understanding of various approaches used to assess petroleum hydrocarbons on the basis of fractions, individual indicator compounds, and appropriate surrogates. This chapter also provides descriptions and evaluations of toxicological studies and epidemiological investigations for these TPH fractions, indicator compounds, and surrogates, and provides conclusions, where possible, on the relevance of toxicity and toxicokinetics data to public health.

6.1.1 TPH Definition and Issues

Overview. The assessment of petroleum hydrocarbon-contaminated sites has involved analysis for "total petroleum hydrocarbons" or TPH. TPH is a loosely defined aggregate that depends on the method of analysis as well as the contaminating material, and represents the total mass of hydrocarbons without identification of individual components (see Chapter 3). As TPH is not a consistent entity, the assessment of health effects and development of health guidance values, such as Minimal Risk Levels (MRLs) for *TPH as a single entity* are problematic. Earlier in the profile (Chapters 2 and 3), various TPH approaches were presented that divide TPH into fractions or groups of compounds based on analytical, fate and transport, and exposure issues. Similarly, several different approaches have also been evolving to assess the health effects of TPH on the basis of indicator compounds for separate fractions, which consist of petroleum hydrocarbons with similar physical and chemical properties. ATSDR's approach to potential health effects from exposure to TPH uses surrogate health effects guidelines for each fraction, whether they represent an individual compound or a whole petroleum product. Additional discussions focusing on these various approaches to health effects assessment are presented in the remainder of this section (6.1). In particular, the ATSDR approach (Section 6.1.3) uses existing ATSDR MRLs for several individual TPH compounds and for specific petroleum products. The use of these MRLs to characterize the health effects of TPH, using an indicator compound and fraction/surrogate approach, is also discussed.

Scope of the Problem. Petroleum hydrocarbons are the principal components in a wide variety of commercial products (e.g., gasoline, fuel oils, lubricating oils, solvents, mineral spirits, mineral oils, and crude oil). Because of widespread use, disposal, and spills, environmental contamination is relatively common. It is important to understand that petroleum products are complex mixtures, typically containing hundreds of compounds. These include various amounts of aliphatic compounds (straight-chain, branched-chain, and cyclic alkanes and alkenes) and aromatic compounds (benzene and alkyl benzenes, naphthalenes, and PAHs). In addition, many petroleum products contain non-hydrocarbon additives such as alcohols, ethers, metals, and other chemicals that may affect the toxicity of the mixture.

The number of individual identified hydrocarbon components of the various petroleum products has been estimated at several hundred to over a thousand. Toxicity data are available for about 95 of these, but only about 25 were considered to have sufficient data to develop toxicity criteria according to the Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG 1997b). ATSDR has derived MRLs for 12 of these compounds (anthracene, benzene, ethylbenzene, fluoranthene, fluorene, *n*-hexane, naphthalene, toluene, *m*-xylene, *p*-xylene, xylenes, and l-methyl naphthalene). EPA has derived Reference Doses (RfDs) and Reference Concentrations (RfCs) for some of the remaining compounds. The TPHCWG (1997c) and the Massachusetts Department of Environmental Protection (MADEP) (Hutcheson et al. 1996) have also derived other health guidance criteria for some of these compounds. Two of these compounds have EPA-derived cancer slope factors and/or unit risks, and a relative potency approach has been developed for some of the PAHs. However, it is not yet possible to assess the overall health implications of TPH from the individual hydrocarbon components because many of the known components lack appropriate, standardized, comparable toxicity data. In addition the cost of analysis for all TPH constituents is usually prohibitive.

Although health effects data are available for some petroleum products, and ATSDR-derived MRLs are available for fuel oil no. 2, JP-4, JP-5/JP-8, JP-7, and kerosene, there are limitations to applying MRLs for the whole products to TPH. A major limitation is that, when released to the environment, the composition of a petroleum product changes due to weathering (i.e., differential fate and transport of its components). Partitioning of fractions consisting of hydrocarbons with similar physical and chemical properties occurs, with migration of some fractions to other locations and environmental media, leaving the relatively nonmobile components (the weathered product) at the original location.

Thus, the actual petroleum hydrocarbon mixture to which a given population is exposed varies with location, time and environmental medium. Accordingly, health effects data for whole petroleum products that are relatively heterogeneous, such as gasoline and JP-4, are not necessarily applicable to the fractions to which exposure actually occurs as a result of transport and weathering. For example, acute inhalation exposure to a fresh spill of gasoline will be to the more volatile constituents, whereas intermediate or chronic oral exposure to drinking water contaminated by a gasoline release will be to the soluble constituents, and exposure to soil at the site of the original spill will be to the less volatile and less soluble constituents. Thus, none of these exposure scenarios would be well represented by experimental data using the whole product.

Additional limitations to the use of health effects data for whole petroleum products include the variable composition of each type of petroleum product due to differences in the crude oil from which it was refined, in the refining processes used, and in the formulation of the final product. Also, non-hydrocarbon additives and contaminants, many of which have significant toxicity, are often included in these whole products (e.g., methyl-*tert*-butyl ether (MTBE) or lead in gasoline). Finally, the identity of the originally released material may not be known or more than one such product may have been released.

Health effects data also are available for some petroleum fractions or process streams that are less heterogeneous. These materials are more representative of the fractions that may partition in the environment and are more useful for assessing health effects of intermediate and chronic exposure to petroleum hydrocarbons. These products are discussed further in Section 6.2. Additional discussion of these and also the more heterogeneous products is presented in Section 6.3.

Mixtures Issues. Petroleum products and their environmental transport fractions are complex mixtures. The preferred method for assessing the health effects of complex mixtures is to use exposure and toxicity data for the mixture of concern, because this approach takes into account toxicological interactions, such as synergism or antagonism, that may occur among the constituents of the mixture. If data for the mixture of concern are not available, then data for a similar mixture may be used. In the absence of pertinent data for the same or a similar mixture, data on the individual components of the mixture are used, taking into account the potential for toxicological interactions. The default assumption, when data regarding interactions are not available or do not clearly indicate

the direction of the interaction, is that the doses or effects are additive (ATSDR 1992; De Rosa et al. 1996; EPA 1986; Johnson and De Rosa 1995; Mumtaz et al. 1994). Other public health aspects of chemical mixtures and TPH have recently been reviewed (Hansen et al. 1998; Todd et al. 1999)

The mixtures of concern for TPH are not the heterogeneous petroleum products, but rather the transport fractions to which populations are more likely to be exposed. Thus, use of health effects data for these fractions would be preferable. When health effects data for petroleum products (mixtures) similar in composition to these fractions are not available, data for individual constituents could be used as surrogates, taking into account the potential for toxicologic interactions. Given the complexity of the interactions data for the individual constituents (Section 6.9) however, the assumption that the toxicity of the constituents is additive may be the most reasonable approach. This implicit assumption underlies the adoption of an MRL as a surrogate value to represent the toxicity of an entire fraction.

6.1.2 Existing Risk-Based Methods for TPH Health Assessment

This section presents approaches of other organizations. The ATSDR approach is presented in Section 6.1.3.

The American Society for Testing and Materials (ASTM) Approach. ASTM (1995) developed a Risk-Based Corrective Action (RBCA) approach for petroleum release sites. Additional information regarding this approach is provided in previous sections of this document and in Chapter 7. The present discussion is limited to health effects aspects of the approach. The RBCA approach is not limited to TPH, but includes any chemical that may be associated with petroleum product releases, including nonhydrocarbon constituents and additives. ASTM used an indicator compound approach that assumes that a significant portion of the total potential impact on human health from all chemicals in a petroleum product spill is due to the indicator compounds, termed chemicals of concern. The ASTM approach assesses the risk of exposure to each chemical of concern separately during the derivation of Tier 1 (general) risk-based screening levels, and Tier 2 and 3 site- specific target levels for contaminated media. Although the use of whole mixture toxicity data and the assumption of additivity for the toxicity of individual chemicals in a mixture were mentioned as options for Tier 2 and 3, neither approach was recommended by ASTM. The criteria to be used in selection of the chemicals of concern for various petroleum products are concentrations in the

product, solubility and mobility, toxicological properties, aesthetic characteristics (e.g., odor), and availability of sufficient information to conduct risk assessments. For gasoline, kerosene, and jet fuels, commonly selected hydrocarbon chemicals of concern are benzene, toluene, ethylbenzene, and xylene (BTEX). Additional chemicals of concern for kerosene and jet fuels are PAHs. For diesel fuel, light fuel oils, and heavy fuel oils, the commonly selected hydrocarbon chemicals of concern are PAHs. Twelve PAHs, including benzo(a)pyrene, were selected for consideration.

The MADEP Approach. The MADEP (Hutcheson et al. 1996; MADEP 1997, 1999) recommends the use of a combination indicator compound and fraction approach for the assessment of health effects from TPH in soil and water as follows:

Carcinogenic Effects. Specific petroleum hydrocarbon indicator compounds that have EPA cancer potency factors are assessed; these are benzene and benzo(a)pyrene. EPA relative potency factors can be used for benz(a)anthracene, indeno(1,2,3-cd)pyrene, dibenz(a,h)anthracene, chrysene, benzo(b)fluoranthene, and benzo(k)fluoranthene.

Noncarcinogenic Effects. The following petroleum hydrocarbon fractions were established based on molecular structure (aromatic versus aliphatic) and then on number of carbon atoms, using toxicologically similar groupings and excluding compounds with less than 5 carbons because their high volatility precludes chronic exposure from spills/releases. With the exception of the aromatic C_5-C_8 fraction, the toxicity of each fraction is represented by the RfD for a representative "reference compound" from the fraction. Analytical methods for these fractions have also been suggested (Section 3.3). Some of these fractions include subfractions that were combined because of similarity of toxicity across fractions or limitations in the toxicity data.

Aromatic fractions

C_5-C_8 , assessed on the basis of the individual indicator compounds-benzene (MADEP RfD derived from inhalation study), toluene, ethylbenzene, and xylenes (EPA RfDi).

C_9-C_{10}, using an EPA RfD for pyrene (the lowest RfD for compounds in this group) as a surrogate and an RfC for xylenes.

$C_{11}C_{12}$, using and EPA RfD for pyrene and an RfC for naphthalene.

Aliphatic fractions

C_5-C_8, using an EPA RfD and RfC for *n*-hexane as a surrogate.

C_9-C_{12}, using a MADEP RfD and RfC for *n*-nonane as a surrogate, based on estimated relative potency of *n*-nonane as compared with *n*-hexane.

C_{13}-C_{18}, using a MADEP RfD and RfC for naphthalene as a surrogate.

$C_{19}C_{35}$,using a MADEP RfD for white mineral oil (but listing eicosane as the reference compound).

The MADEP (1997) has published a draft report for public comment regarding implementation of their approach. This report references the TPHCWG (1997a, 1997b, 1997c) approach (below), particularly in defining fractions with regard to transport properties, which are related to the equivalent (or relative) carbon number indexes for the compounds.

The TPHCWG Approach. The TPHCWG (1997a, 1997b, 1997c) also recommends a combination indicator compound and fraction approach for TPH, but it differs from the MADEP approach in the elimination of assessment for noncarcinogenic effects if carcinogens are present above regulatory criteria, in the basis for selection of the fractions, and in a more extensive use of toxicity data for mixtures to represent the toxicity of the fraction. Some petroleum hydrocarbon fractions listed below include subfractions that were combined because of similarity of toxicity across fractions or limitations in the toxicity data.

Carcinogenic Effects. Specific petroleum hydrocarbon indicator compounds that have EPA cancer potency factors are assessed (i.e., benzene and benzo(a)pyrene).

Noncarcinogenic Effects. These effects are assessed only if the carcinogenic indicator compounds are not detected or are below regulatory criteria. The following petroleum hydrocarbon fractions, minus the carcinogenic indicator compounds, were selected as representing compounds with similar transport properties. Toxicity values for constituents of the fraction or for a similar mixture were selected to represent the toxicity of the fraction. Aromatic and aliphatic hydrocarbons are considered separately and further subdivided on the basis of equivalent carbon number index (EC). This index is equivalent to the retention time of the compounds on a boiling point GC column (non-polar capillary column), normalized to the *n*-alkanes. Physical and chemical properties of hydrocarbons that are

useful in predicting transport (vapor pressure, solubility, partition coefficient, Henry's law constants) are predictably related to the EC and can be estimated using algorithms (see Chapter 5).

Aromatic fractions

EC_5-EC_8, using EPA RfD and RfC for toluene as a surrogate.

$EC_{>8}$-EC_{16}, using EPA RfDs (all the same value) for two compounds (cumene [isopropylbenzene] and naphthalene) as a surrogate and an RfC for C_9 aromatics (hi-flash aromatic naphtha).

$EC_{>16}EC_{35}$, using the EPA RfD for pyrene (C_{16}) as a surrogate. Anthracene, fluorene, and fluoranthene are also in this group; however, pyrene was selected because it had the lowest RfD.

Aliphatic fractions

EC_5-EC_8, using TPHCWG RfD (derived from inhalation data) as a surrogate and RfC for commercial hexane, a mixture of C_6 hydrocarbons containing 53% *n*-hexane.

$EC_{>8}$-EC_{16}, using TPHCWG RfD and RfC for dearomatized petroleum streams (white spirit).

$EC_{>14}$-EC_{35}, using TPHCWG RfD for white mineral oils.

The MADEP (Hutcheson et al. 1996; MADEP 1997) and the TPHCWG (1997a, 1997b, 1997c) approaches both assume additivity of the indicator compounds and the hydrocarbon fractions in assessing the potential for adverse effects of TPH on health. In contrast, the ASTM approach ten to assess each individual TPH indicator chemical separately and without regard to the presence of other petroleum hydrocarbons and the potential for additivity or interactions, although it does not preclude a consideration of these factors.

6.1.3 Overview of the ATSDR Approach

In formulating an approach to health assessment of TPH, ATSDR has drawn on the experience of other groups that have been developing approaches to health-based assessment for TPH (i.e., ASTM [1995]; Hutcheson et al. [1996]; and TPHCWG [1997a, 1997b, 1997c]), but has developed an approach designed to address its own specific concerns and mandates. A notable difference between ATSDR and these other groups is that the other groups have focused on longer-term exposure

scenarios, whereas ATSDR is concerned with the entire spectrum of possible exposure periods from acute through chronic. In addition, the health guidance values developed by ATSDR, MRLs, are intended to serve as screening levels by ATSDR health assessors to identify contaminants and potential health effects that may be of concern at hazardous waste sites. **MRLs are not intended to define clean-up or action levels.**

The ATSDR approach, as reflected in this profile, focuses on an assessment of the health effects of petroleum hydrocarbon transport fractions, as suggested by the TPHCWG (1997a, 1997b, 1997c). This approach is the most universally useful, given the limitations to using data for the whole petroleum products or individual constituents, discussed in Chapter 2 and in Section 6.2.1 above. Methods of analysis for these fractions are available, and modeling can be performed to predict exposure to the fractions. The assessment of the health effects of the fractions by ATSDR is similar but not identical to that of the TPHCWG. In addition, to capitalize on the best features of the MADEP (Hutcheson et al. 1996) and TPHCWG (1997a, 1997b, 1997c) approaches, the aromatic EC_5-EC_8 fraction has been redefined as an EC_5-EC_9 fraction, so that it includes all the BTEXs. The aromatic $EC_{>8}$-EC_{16} fraction is then redefined as an $EC_{>9}$-EC_{16} fraction.

Carcinogenic Effects. Specific hydrocarbon indicator compounds that have EPA cancer risk estimates are assessed; these are benzene and benzo(a)pyrene. EPA relative potency factors can be used for benz(a)anthracene, indeno(1,2,3-cd)pyrene, dibenz(a,h)anthracene, chrysene, benzo(b)fluoranthene, and benzo(k)fluoranthene.

Noncarcinogenic Effects. The following petroleum hydrocarbon fractions, including the carcinogenic indicator compounds, were selected as representing compounds with similar transport properties, based on the recommendations of the TPHCWG (1997b, 1997c), with an adjustment of the lower EC aromatic fractions in order to include all the BTEXs in the first fraction, as discussed above. As with the MADEP and TPHCWG approaches, some of the fractions include subfractions that have been combined because of similarity of health effects across fractions or limitations in the health effects data. Provisional recommendations regarding suitable MRLs are made, using a surrogate approach as needed and appropriate. The MRL for the surrogate compound or for a petroleum product similar in composition to the fraction is used to indicate the potential toxicity of the entire mass of the fraction.

Aromatic Fractions

EC_5-EC_9, using inhalation and oral MRLs specific to each individual indicator compound-benzene, toluene, ethylbenzene, and the xylenes.

$EC_{>9}$-EC_{16}, using a chronic inhalation MRL and acute and intermediate oral MRLs for naphthalene as surrogates.

EC_{16}-EC_{35}, using an intermediate oral MRL for fluorene and fluoranthene as a surrogate.

Aliphatic Fractions

EC_5-EC_8, using a chronic inhalation MRL for *n*-hexane as a surrogate.

$EC_{>8}$-EC_{16}, using a chronic inhalation MRL for JP-7.

$EC_{>16}$-$EC_{>35}$, using health effects data for mineral oils, but no MRLs are available.

The health effects of these fractions are discussed in Section 6.2, and details of the selection of the fraction-specific MRLs can be found in Section 6.6. These fraction-specific values are provisional values, reflecting the uncertainty inherent in this approach, as discussed in Section 6.6. Further information on ATSDR MRLs is given in Appendix A, while information on other toxicity criteria such as RfDs and RfCs, is provided in Chapter 7.

ATSDR has already prepared toxicological profiles on a large number of individual constituents of TPH and on a number of whole petroleum products. In order to give an overall perspective on the toxicology of TPH, without duplicating the existing profiles, this toxicological profile will present brief summaries of the health effects of these individual petroleum hydrocarbon compounds and petroleum products. MRLs have been derived for a number of these compounds, which serve as indicator and surrogate compounds for the ATSDR approach as outlined above. Thus, consideration of these compounds as part of the TPH contamination profile is useful. Similarly, information regarding the extent and identity of petroleum product contamination may be available, and toxicity information and MRLs for these original products may be useful in some circumstances for assessing potential health effects. These brief summaries of information on the individual compounds and on petroleum products that are representative of particular fractions occur during the discussion of the health effects of the fractions in Section 6.2. Information on petroleum products, including the more heterogenous mixtures, also is presented in Section 6.3. The reader is encouraged to consult the original toxicological profiles listed in Appendix A and other cited sources for more detail.

The content of this chapter and this document is different from that of a standard toxicological profile, in recognition of the extensive assessments of individual petroleum hydrocarbons already performed by ATSDR and other agencies, and the need for an approach that focuses on the most important information. This chapter presents the ATSDR perspective and approach, and serves as a guide to sources of more detailed information.

6.2 DISCUSSION OF HEALTH EFFECTS BY FRACTION AND ROUTE OF EXPOSURE

Because of the complexity of TPH, and the existence of extensive ATSDR and TPHCWG documentation for constituents of TPH and for petroleum products and mixtures corresponding to some of the fractions, this section of the document adopts a "handbook approach" to delineating the health effects of TPH. The organization and content of this section, while retaining an emphasis on route and duration of exposure and on type of health effect, is streamlined in order to avoid duplication of existing resources and to help public health professionals, and others who address the needs of people living or working near hazardous waste sites, to gain an understanding of the characteristic health effects of TPH fractions. The juxtaposition of information on fraction composition with information on health effects for fraction constituents facilitates evaluation of the suitability of the existing health effects information to represent the potential health effects of the entire fraction. Further discussion of the suitability and representativeness of the information is presented in Section 6.6.

Thus, for each fraction, the components of the fraction are delineated first. Health effects for the fraction are then discussed by route of exposure. This discussion includes information on individual constituents of the fraction and on mixtures that correspond to the fraction. The text focuses on the major, sensitive, and/or characteristic end points.

The figures give a *condensed picture* of exposure-effect relationships for each fraction. They show the lowest reliable lowest-observed-adverse-effect-level (LOAEL) in animals and humans for each route, exposure period, and end point, including cancer. The three exposure periods-acute (14 days or less), intermediate (15-365 days), and chronic (365 days or more)-are represented. Different symbols are used to represent different compounds or mixtures, with open symbols for animals and

closed for humans. For additional information, including no-observed-adverse-effect levels (NOAELs), classification of LOAELs into “less serious” or “serious” effects, and details of the actual studies, the reader is encouraged to consult the sources referenced in the figures. Because cancer effects could occur at lower exposure levels than the exposures plotted in some of the figures, these figures also show a range for the upper bound of estimated excess risks, ranging from an estimate of 1 in 10,000 to 1 in 10,000,000 ($l0^{-4}$ to 10^{-7}), as developed by EPA.

In addition, estimates of minimal risk to humans (MRLs) are plotted. An MRL is defined as an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (noncarcinogenic) over a specified duration of exposure. MRLs are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration within a given route of exposure. MRLs are based on noncancerous health effects only and do not consider carcinogenic effects. MRLs can be derived for acute, intermediate, and chronic duration exposures for inhalation and oral routes. Appropriate methodology does not exist to develop MRLs for dermal exposure.

Although methods have been established to derive these levels (Barnes and Dourson 1988; EPA 199Oc), uncertainties are associated with these techniques. Furthermore, ATSDR acknowledges additional uncertainties inherent in the application of the procedures to derive less than lifetime MRLs. As an example, acute inhalation MRLs may not be protective for health effects that are delayed in development or are acquired following repeated acute insults, such as hypersensitivity reactions, asthma, or chronic bronchitis. As these kinds of health effects data become available and methods to assess levels of significant human exposure improve, these MRLs will be revised.

The figures in this section were compiled primarily from the tables and figures showing *Levels of Significant Exposure* in ATSDR toxicological profiles. To fill data gaps for some of the fractions, pertinent additional health effects information from EPA sources and from the TPHCWG (1997c) was included. MADEP also was consulted, but did not appear to provide significant additional information for this purpose. (RfCs and RfDs from these sources are reported in Chapter 7.)

6.2.1 Aromatic EC_5-EC_9 Indicator Compounds

This fraction consists of indicator compounds: benzene, toluene, ethylbenzene, and xylene (mixture and individual isomers *o*-, *m*-, *p*-). These indicator compounds are often referred to as the BTEXs, and are commonly assessed using MRLs (or EPA toxicity values) specific to each compound. Styrene also would fall in this fraction, but does not appear to be a significant constituent of the petroleum products whose composition was reported by TPHCWG (1997c). The BTEXs are the subject of separate ATSDR toxicological profiles (ATSDR 1994, 1995b, 1997a, 1999a); these profiles should be consulted for detailed information on these compounds. The information in Sections 6.2.1.1 through 6.2.1.3 is taken from these profiles; for the sake of readability, references to these ATSDR profiles will not be repeated in these sections.

6.2.1.1 Inhalation Exposure

All the BTEXs cause neurological effects. Neurological effects are the basis for MRLs for both acute and chronic exposures to toluene and mixed xylenes, and for intermediate exposures to benzene; neurological effects are not as sensitive for ethylbenzene. The neurological effects consist primarily of central nervous system depression. Toluene's neurotoxicity also includes ototoxicity. Evidence of hearing loss has been seen in both occupationally exposed humans and in animals. There is limited evidence that chronic inhalation exposure to benzene may affect the peripheral nervous system; this evidence is from a single study of occupationally exposed humans who also had aplastic anemia.

Benzene is the only BTEX that has well characterized hematological, immunological, and lymphoreticular effects in humans and animals at low levels of inhalation exposure. Immunological and lymphoreticular effects are the basis for the derivation of the acute inhalation MRL for benzene. Benzene affects hematopoiesis, decreasing the production of all major types of blood cells, and can also cause hyperplasia.

Developmental effects are the basis for intermediate MRLs for ethylbenzene and mixed xylene, indicating that the embryo/fetus may be particularly sensitive to these two BTEXs.

Benzene is considered to be carcinogenic to humans by the inhalation route of exposure (EPA weight-of-evidence Group A, human carcinogen). Occupational exposure to benzene was associated with

increased incidences of nonlymphocytic leukemia. Studies in animals also found increased incidences of neoplasia in animals treated by inhalation or gavage with benzene.

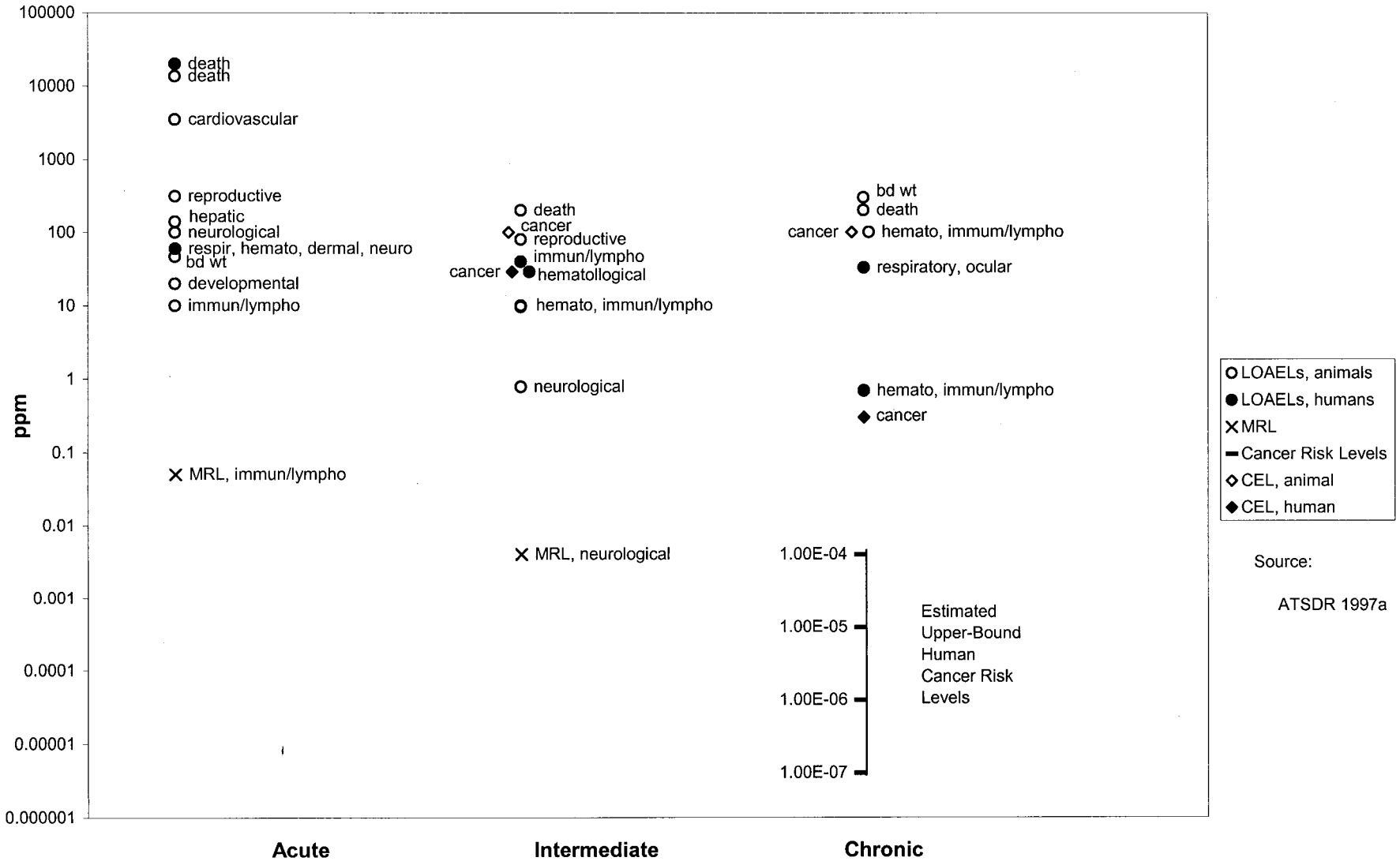
Although ethylbenzene was classified in EPA weight-of-evidence Group D (not classifiable as to human carcinogenicity), subsequent publication of a chronic inhalation study of ethylbenzene provides evidence of carcinogenicity in rats and mice, and indicates a need for reassessment. Toluene and mixed xylene are classified in Group D.

The lowest reliable LOAEL values for the BTEXs are summarized in Figure is 6-l through 6-4, as are MRLs and cancer risk levels. The data for each compound are presented in a separate figure because of the voluminous data available for each and because these compounds are commonly assessed using the exposure data and MRLs (or EPA toxicity values) specific for each. The data for mixed xylene are extensive, and MRLs are available for all three durations, whereas little data and no MRLs are available for the individual isomers (*o*-, ***m*-,** and *p*-). The inhalation toxicity data for the individual isomers are reasonably similar to those for the mixture. Accordingly, only the data for mixed xylene are included in the figure. More detailed information is available in the ATSDR toxicological profiles on the individual compounds (ATSDR 1994, 1995d, 1997a, 1999a), from which the information in this section is drawn.

6.2.1.2 Oral Exposure

Data for the oral route of exposure are less extensive. The BTEXs cause neurological effects, generally central nervous system depression, by the oral route. This is a sensitive effect for toluene and *p*-xylene, for which it is the basis of acute and/or intermediate MRLs. Renal and hepatic effects are also seen with oral exposure to these compounds. Renal effects are the basis for the intermediate MRL for mixed xylenes and hepatic effects are the basis for the intermediate MRL for *m*-xylene. The hepatic effects tend to be mild, including increased liver weight and cytochromes P-450 and b5 contents. Benzene causes hematological effects by the oral route that are similar to those seen from inhalation exposure.

Figure 6-1. Aromatic EC_5-EC_9 Exposures Associated with Health Effects - Inhalation - Benzene



Source:

ATSDR 1997a

Figure 6-2. Aromatic EC_5-EC_9 Exposures Associated with Health Effects - Inhalation - Toluene

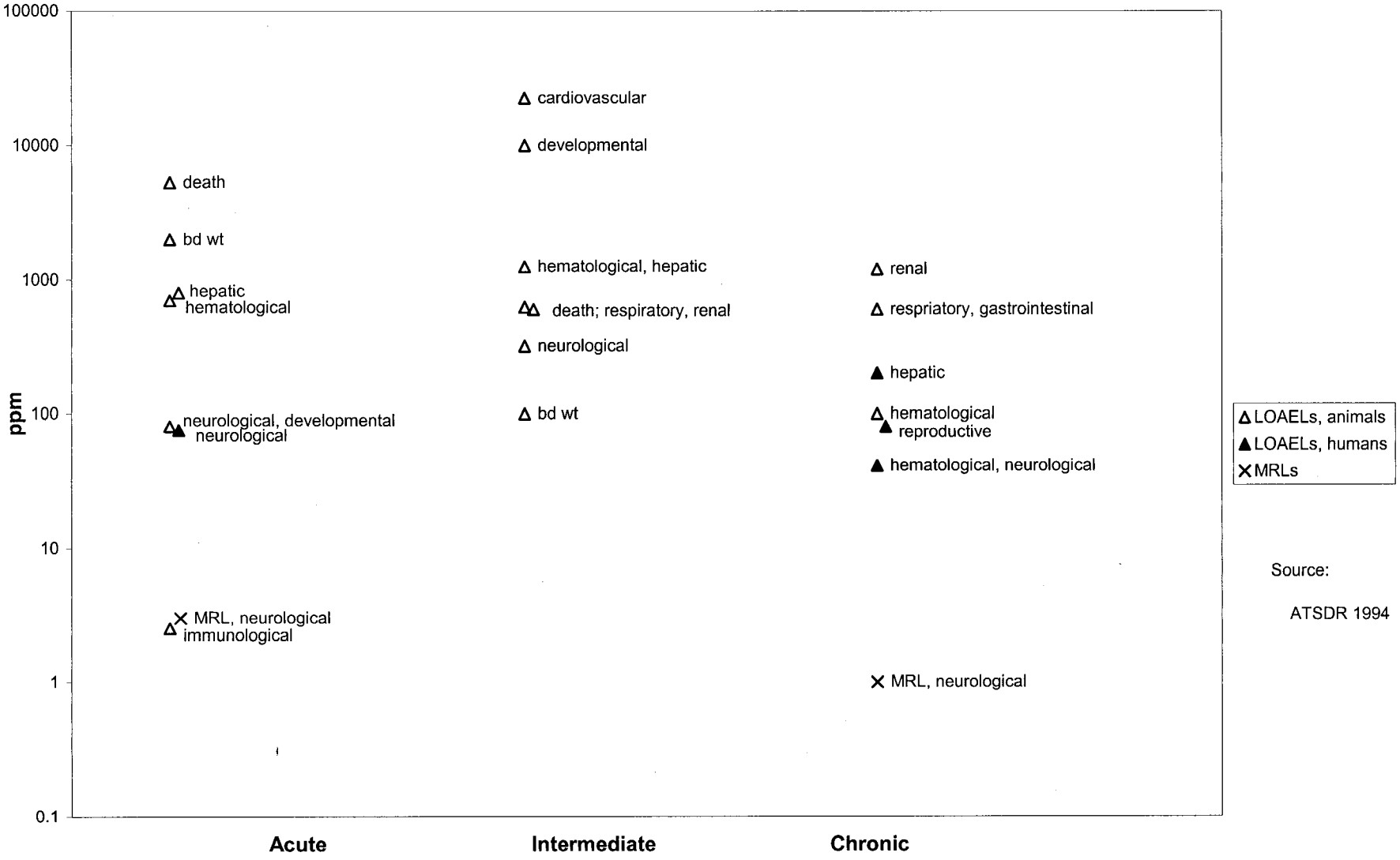


Figure 6-3. Aromatic EC_5-EC_9 Exposures Associated with Health Effects - Inhalation - Ethylbenzene

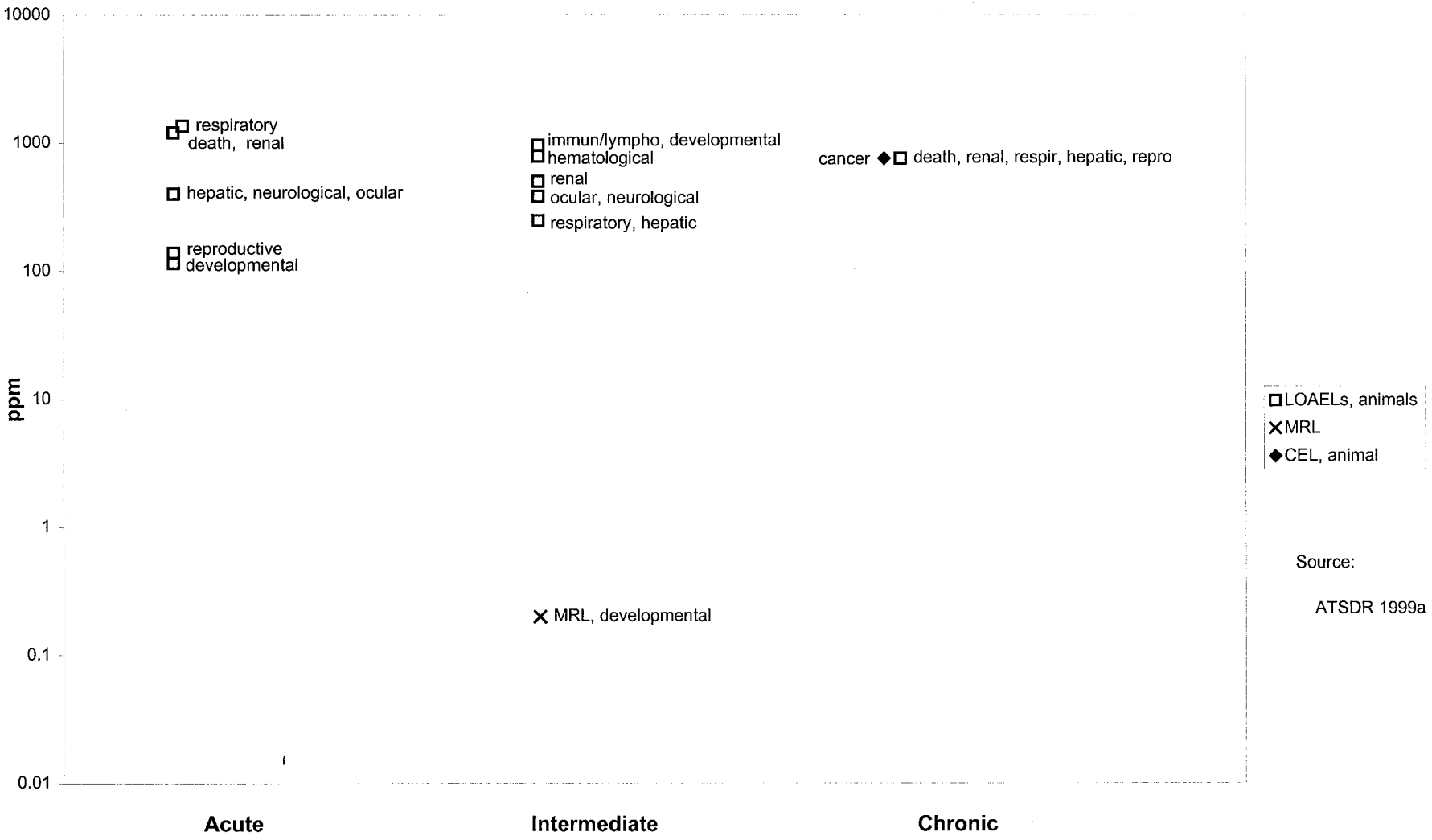
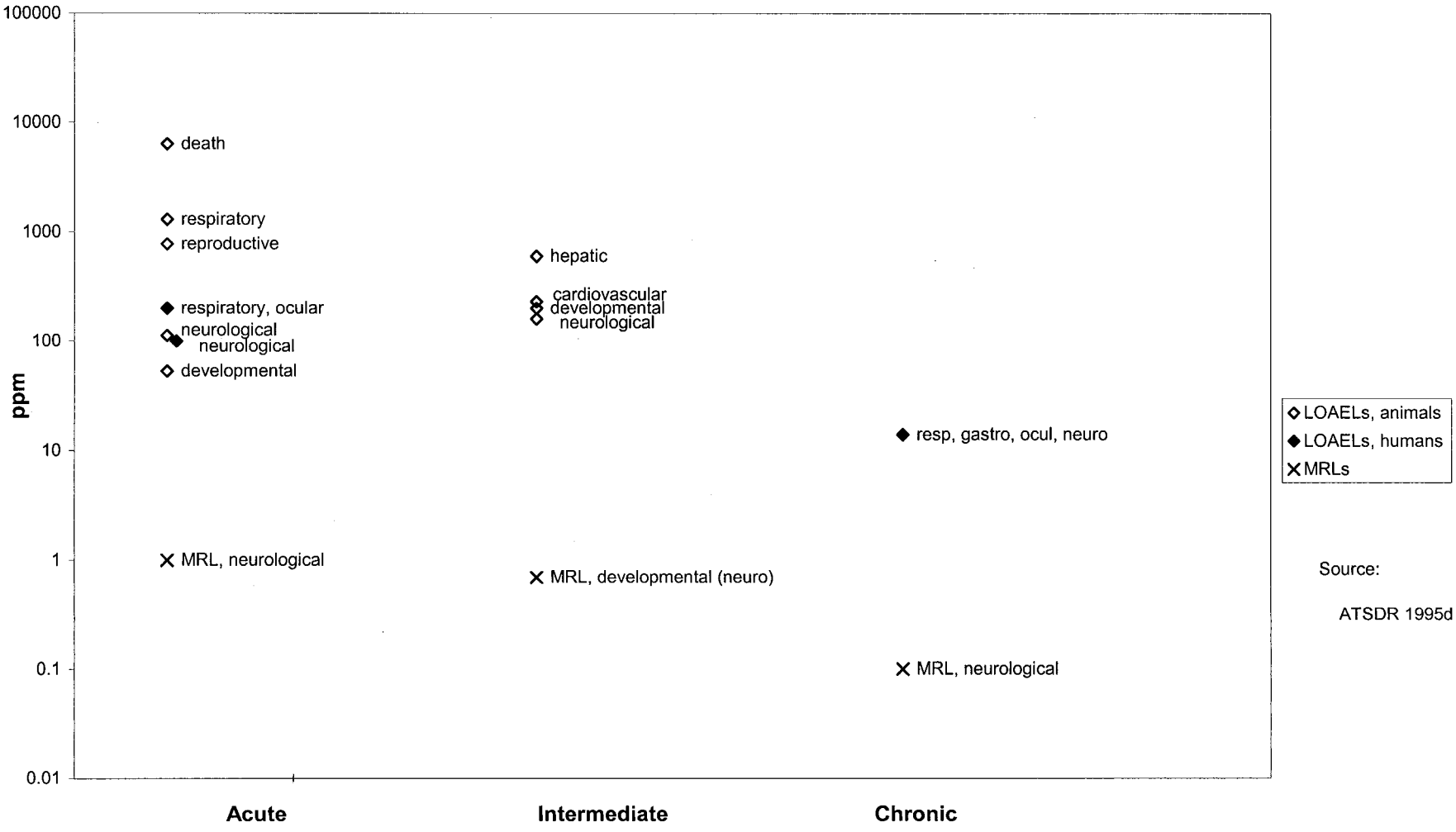


Figure 6-4. Aromatic EC_5-EC_9 Exposures Associated with Health Effects - Inhalation - Mixed Xylene



Source:

ATSDR 1995d

Benzene is considered to be carcinogenic (EPA weight-of-evidence Group A) to humans by either inhalation or oral exposure, based on occupational studies that showed increased incidences of nonlymphocytic leukemia in humans exposed by inhalation, with supporting data from oral and inhalation studies in animals. Results of a recently published study of ethylbenzene in animals indicate carcinogenicity by the inhalation route, but there is no evidence of carcinogenicity by the oral route. Toluene and mixed xylene are classified in EPA weight of evidence Group D (not classifiable as to human carcinogenicity).

The lowest reliable LOAEL values for the BTEXs are summarized in Figures 6-5 through 6-7, as are MRLs and cancer risk levels. With the exception of ethylbenzene, the data for each of the BTEXs are presented in a separate figure because of the voluminous data available for each and because these compounds are commonly assessed using the exposure data and MRLs (or EPA toxicity values) specific for each. There are only two pertinent LOAELs and no MRLs for ethylbenzene, so the LOAELs for ethylbenzene are plotted with those for toluene, and indicated by a different symbol. More detailed information is available in the ATSDR toxicological profiles on the individual compounds (ATSDR 1994, 1995d, 1997a, 1999a), from which the information in this section is drawn.

6.2.1.3 Dermal Exposure

Information on the health effects of dermal exposure to the BTEXs is limited. Skin and eye irritation are well documented, but effects from systemic absorption are not. ATSDR (1997a) concluded that it is reasonable to expect that adverse hematological and immunological effects might occur following dermal exposure to benzene, because benzene is absorbed through the skin and absorption through any route would increase the risk of these effects. For more detailed information, see the ATSDR toxicological profiles on the individual compounds (ATSDR 1994, 1995d, 1997a, 1999a), from which the information in this section is drawn.

6.2.2 Aromatic $EC_{>9}$-EC_{16} Combined Fractions

$EC_{>9}$-EC_{10} fraction: includes cumene (isopropylbenzene), *n*-propylbenzene, the methyl-ethylbenzenes, some trimethylbenzene isomers, and the branched-chain butylbenzenes. None of these compounds is the subject of an ATSDR toxicological profile.

Figure 6-5. Aromatic EC_5-EC_9 Exposures Associated with Health Effects - Benzene - Oral

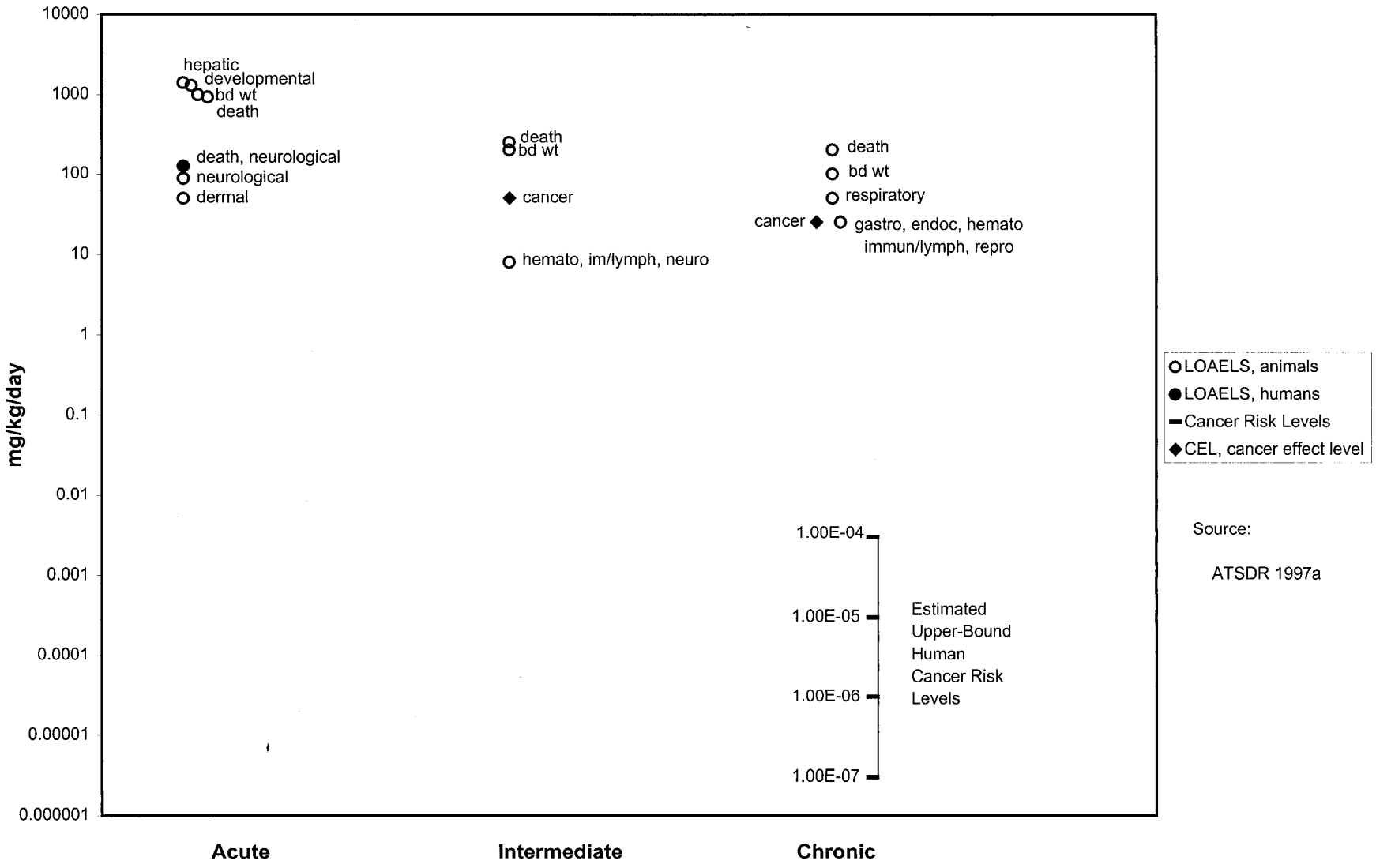
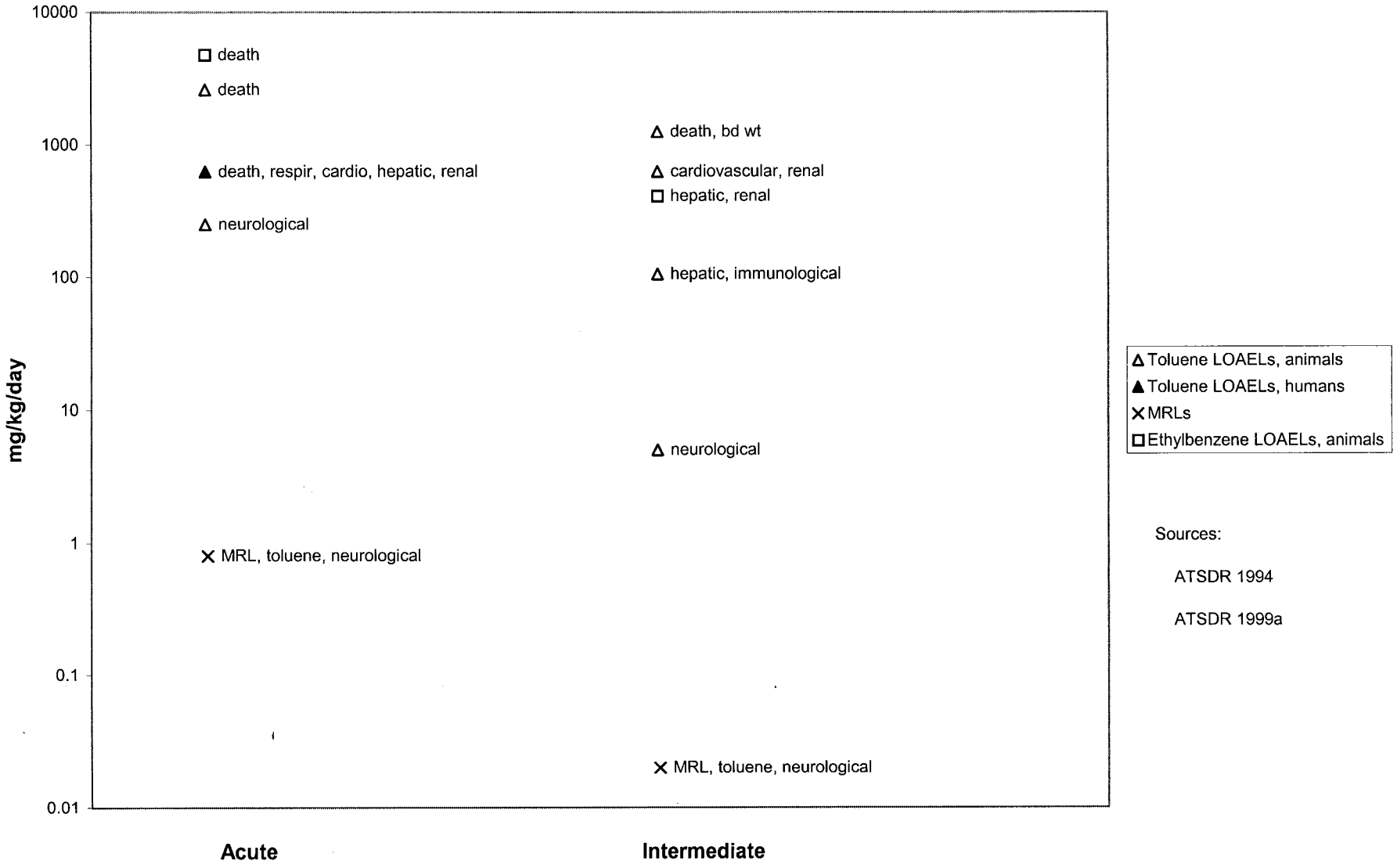


Figure 6-6. Aromatic EC_5-EC_9 Exposures Associated with Health Effects - Oral - Toluene and Ethylbenzene

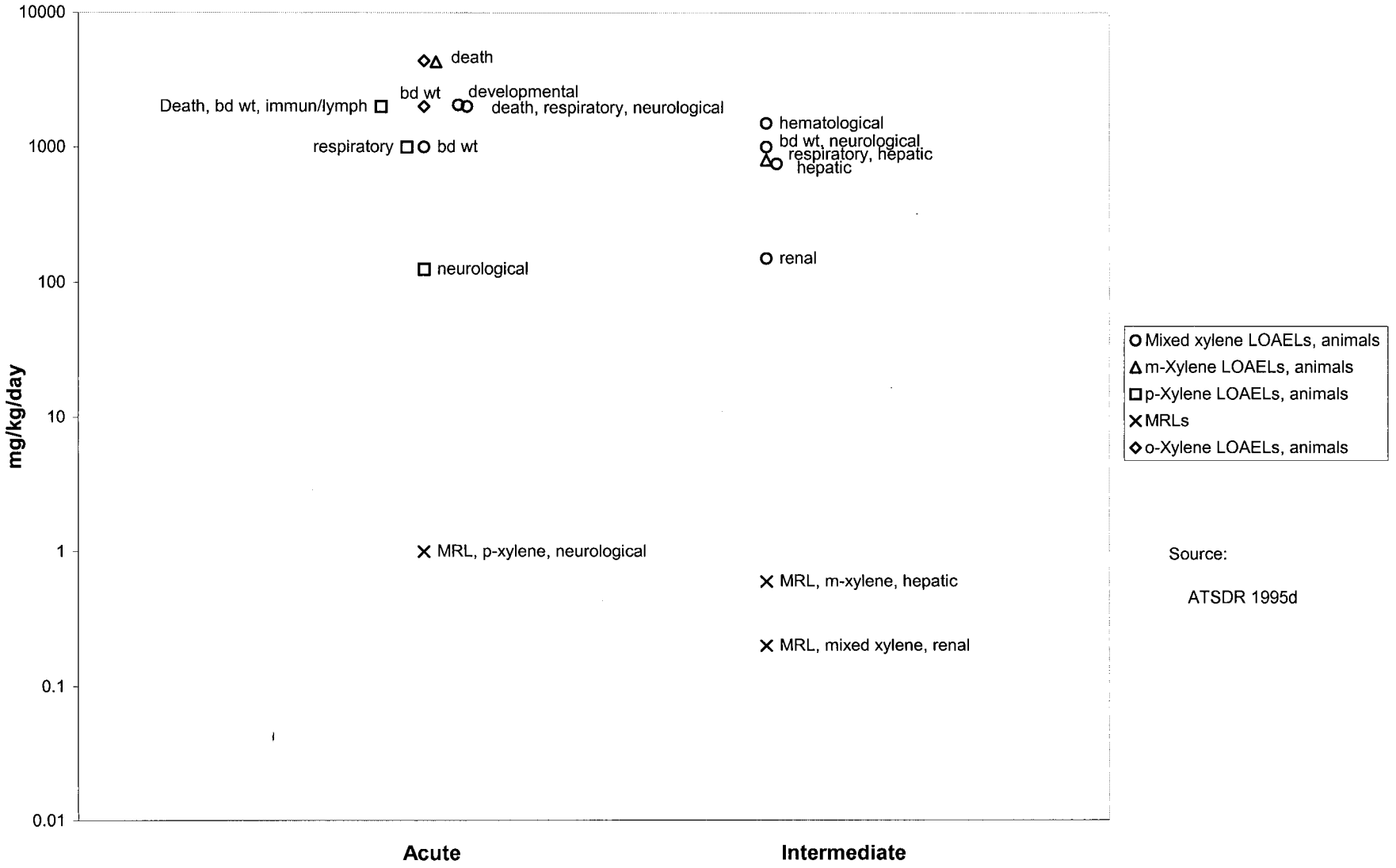


Sources:

ATSDR 1994

ATSDR 1999a

Figure 6-7. Aromatic EC_5-EC_9 Exposures Associated with Health Effects - Oral - Xylenes



Source:

ATSDR 1995d

$EC_{>10}$-EC_{12} fraction: includes *n*-butyl and *n*-pentylbenzene, a trimethylbenzene isomer and various other multi-substituted alkylbenzenes, as well as indan, methylindans and naphthalene. The only compound in this fraction for which an ATSDR toxicological profile is available is naphthalene (ATSDR 1995e).

$EC_{>12}$- EC_{16} fraction: includes a few longer-chain and multi-substituted alkyl benzenes, biphenyls, the mono- and dimethylnaphthalenes, and PAHs, including acenaphthene and acenaphthylene. The monomethylnaphthalenes (l- and 2-methyl naphthalene) are discussed in the ATSDR toxicological profile on naphthalene (ATSDR 1995e) and acenaphthene and acenaphthylene are included in the ATSDR toxicological profile on PAHs (ATSDR 1995f).

6.2.2.1 Inhalation Exposure

No toxicological profiles are available for petroleum hydrocarbons in the $EC_{>9}$-EC_{10} fraction. Inhalation exposure to isopropylbenzene (cumene) and to the trimethylbenzene is known to have neurological and respiratory irritant effects (EPA 1997a, 1998b; TPHCWG 1997c), but these may not be the most sensitive effects of inhalation exposure to the compounds in this fraction. EPA (1998b) concluded that the critical effect of inhalation exposure to isopropylbenzene was increased renal weights in female rats and increased adrenal weights in both sexes of rats in a 13-week inhalation study (Cushman et al. 1995). An RfC was based on these data. Toxicity data for a mixture of C_9 aromatics, consisting primarily of trimethylbenzene and methylethylbenzene isomers, have been assessed (as the basis for an RfC) by the TPHCWG (1997c). The critical effects were hepatic and renal.

Hemolytic anemia is a frequent consequence of acute inhalation exposure to naphthalene in humans, particularly infants and those with a G6PD genetic defect. Exposure-effect relationships for hemolytic anemia are not well characterized. Ocular effects, including cataracts, have been reported in humans exposed to naphthalene vapors, but exposure levels were not known. In mice, respiratory effects are a sensitive effect of inhalation exposure to naphthalene. A chronic MRL has been derived for naphthalene based on respiratory effects in mice-chronic inflammation and regeneration of the nasal epithelium and inflammation of the lung epithelium. In addition, the same study in mice reported an increased incidence of lung adenomas in female but not in male mice (ATSDR 1995e).

The EPA classified naphthalene in Group D (not classifiable as to human carcinogenicity) prior to publication of this study, but notes that naphthalene may be more appropriately classified in Group C (possible human carcinogen) (EPA 1998b).

No MRLs have been developed for compounds in the $EC_{>9}$ –EC_{16} fraction. Only acenaphthylene has been assessed by the EPA for carcinogenicity; the data were considered inadequate (Group D) (ATSDR 1995e, 1995f).

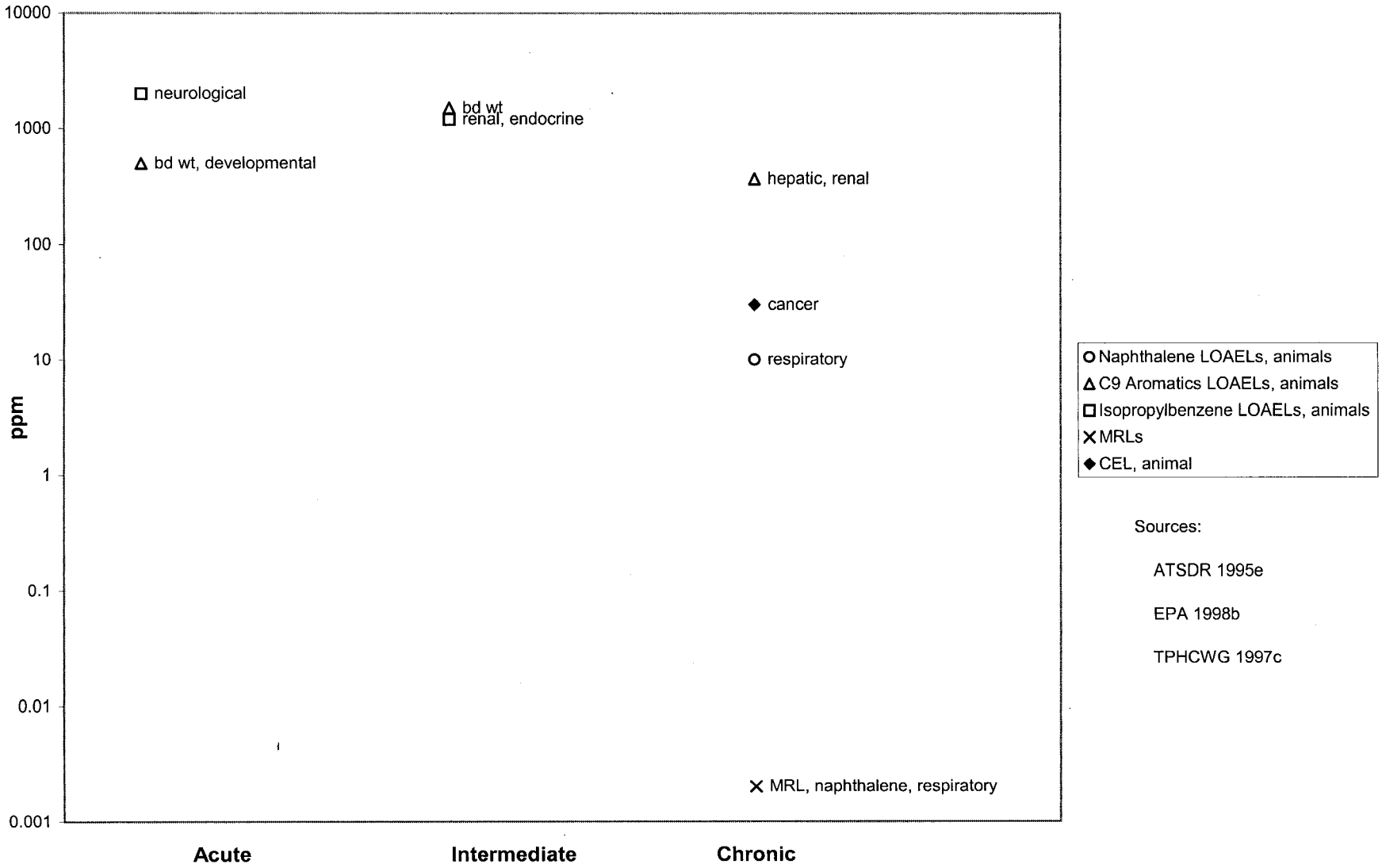
The lowest reliable LOAEL values for the combined aromatic $EC_{>9}$-EC_{16} fraction are summarized in Figure 6-8, as are MRLs. Because so few of the compounds in this fraction have been assessed by ATSDR, additional information from EPA sources and the TPHCWG (1997c) have been added. More detailed information is available in the ATSDR toxicological profiles on the individual compounds and in the other sources noted above.

6.2.2.2 Oral Exposure

There are no toxicological profiles or MRLs for compounds in the $EC_{>9}$-EC_{10} fraction. Toxicity data, primarily from subchronic oral studies in rats, have been assessed by EPA during the derivation of RfDs for two of the compounds-isopropylbenzene (cumene) (EPA 1997a) and 1,3,5-trimethylbenzene (EPA 1996). The critical effect for isopropylbenzene was renal; for 1,3,5trimethylbenzene, the critical effect was a combination of renal, hepatic, and other systemic effects. Oral data for these compounds were limited. Isopropylbenzene has been classified in Group D (not classifiable as to human carcinogenicity) (EPA 1998b). 1,3,5-Trimethylbenzene has not been classified and does not appear to have been studied for carcinogenicity.

Naphthalene, a constituent of the $EC_{>10}$-EC_{12} fraction, produces hemolytic anemia in humans when ingested. As mentioned previously, individuals with a genetic G6PD deficiency have an increased susceptibility to this effect. Little dose-effect information is available for this effect in humans or in animals; dogs appear to be more susceptible than other animal species. Ocular effects occur with high-dose oral administration of naphthalene in animals. The most common effect is cataract formation, but retinal damage has also been noted (ATSDR 1995e). More sensitive effects in animals are neurological effects (central nervous system depression in pregnant animals) and mild hepatic

Figure 6-8. Aromatic $EC_{>9}$-EC_{16} Exposures Associated with Health Effects - Inhalation



Sources:

ATSDR 1995e

EPA 1998b

TPHCWG 1997c

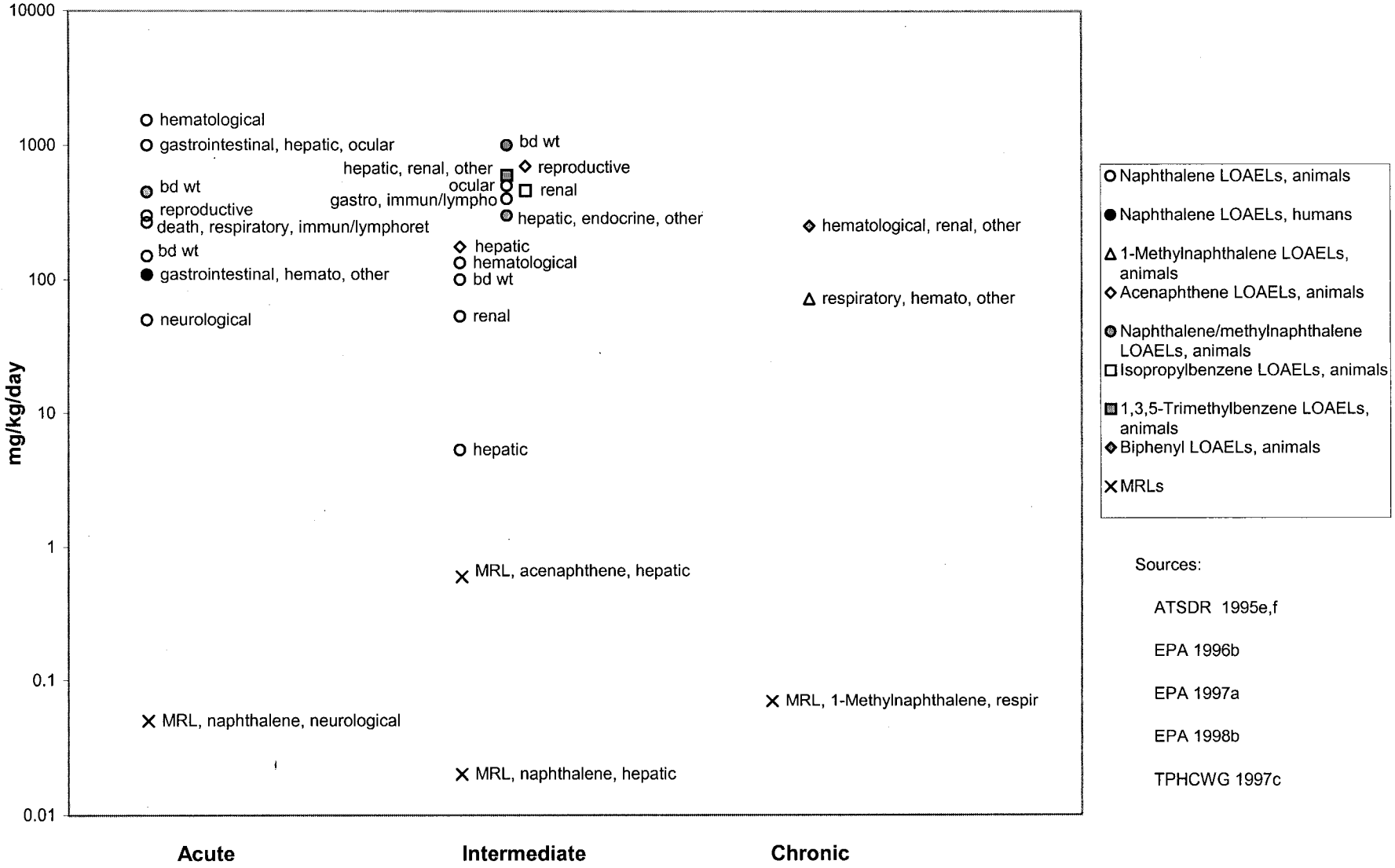
effects (altered microsomal enzyme activities and blood chemistry findings). The EPA classified naphthalene in Group D (not classifiable as to human carcinogenicity) prior to publication of an inhalation study that reported an increased incidence of pulmonary adenomas in female mice, but notes that naphthalene may be more appropriately classified in Group C (possible human carcinogen) (EPA 1998b). No studies documenting carcinogenic effects by the oral route were found (ATSDR 1995e).

Some of the constituents of the $EC_{>12}$-EC_{16} fraction have been evaluated in ATSDR toxicological profiles. Although the database for l-methyl naphthalene is very limited, it includes a chronic study in mice, which serves as the basis for a MRL (ATSDR 1995e). The only effects seen were respiratory (nodular alveolar proteinosis) and hematological (slight increases in hemoglobin parameters and elevated monocyte counts). The limited database for acenaphthene indicates that hepatic effects may be a sensitive consequence of intermediate exposure in mice; the intermediate MRL was based on this finding (ATSDR 1995f). Biphenyl, not included in an ATSDR toxicological profile, has been evaluated by EPA (1998b), which derived an RfD based on renal effects in a chronic study in rats. Hematological effects (reduced hemoglobin), decreased food intake, and decreased longevity also occurred, but renal effects appeared more sensitive. Although the database for this compound is limited, it indicates that reproductive and developmental end points are not as sensitive as renal.

Biphenyl (EPA 1998b) and acenaphthylene (ATSDR 199X) have been classified in Group D (not classifiable as to human carcinogenicity).

The lowest reliable LOAEL values and the available MRLs for the combined aromatic $EC_{>9}$-EC_{16} fraction are summarized in Figure 6-9. Because only a few of the compounds in this fraction have been assessed by ATSDR, additional information from EPA sources and the TPHCWG (1997c) has been added. More detailed information is available in the ATSDR toxicological profiles on the individual compounds and in the other sources noted above.

Figure 6-9. Aromatic $EC_{>9}$-EC_{16} Exposures Associated with Health Effects - Oral



Sources:

ATSDR 1995e,f

EPA 1996b

EPA 1997a

EPA 1998b

TPHCWG 1997c

6.2.2.3 Dermal Exposure

The compounds in the combined $EC_{>9}$-EC_{16} fraction are known to be irritating to the skin, but little information is available to suggest systemic toxicity from dermal exposure alone. Naphthalene, however, has caused hematological effects in human infants exposed to diapers that had been treated with naphthalene moth balls (ATSDR 1995e).

6.2.3 Aromatic $EC_{>16}$- EC_{35} Combined Fractions

This fraction consists entirely of PAHs. The more environmentally and toxicologically significant PAHs are the subjects of the ATSDR toxicological profile on PAHs (ATSDR 1995f); two of these PAHs, acenaphthene and acenaphthylene, are constituents of the $EC_{>12}$-EC_{16} fraction, discussed previously, and the remaining 15 are constituents of the $EC_{>16}$- EC_{35} combined fraction, described below.

$EC_{>16}$- EC_{21} fraction: includes anthracene, fluorene, phenanthrene and pyrene, which are discussed in ATSDR (1995f), and other, less well known PAHs such as substituted fluorenes, anthracenes, and phenanthrenes.

$EC_{>21}$- EC_{35} fraction: includes benz(a)anthracene; benzo(b)-, benzo(j)-, and benzo(k)fluoranthene; benzo(g,h,i)perylene; benzo(a)- and benzo(e)pyrene; chrysene; dibenz(a,h)anthracene; fluoranthene; and indeno(1,2,3-c,d)pyrene, which are discussed in ATSDR (1995f), as well as other, less well known PAHs, that include substituted pyrenes, fluorenes, and fluoranthenes.

6.2.3.1 Inhalation Exposure

Little information regarding the inhalation toxicity of PAHs in the $EC_{>16}$-EC_{35} combined fraction is available, and no inhalation MRLs have been derived. A 4-week study of nose-only inhalation exposure of rats to an aerosol of benzo(a)pyrene identified no treatment-related lesions in the respiratory tract or the kidneys at the single exposure level tested. Respiratory effects, including reduced lung function and abnormal chest X-ray, have been seen in humans exposed occupationally to benzo(a)pyrene and particulate matter. Hamsters exposed by inhalation of benzo(a)pyrene particles developed respiratory tract tumors (nasal, pharyngeal, laryngeal, and tracheal) (ATSDR 1995f).

Assessments of carcinogenicity by EPA have placed some of these compounds in EPA Weight-of-Evidence Group B2 (probable human carcinogen) and others in D (not classifiable as to human carcinogenicity). These classifications were based on evidence from dermal and parenteral studies, and for a few PAHs, oral and inhalation studies, all in animals. See Section 6.2.3.2 and Section 6-6 for specific information regarding EPA cancer assessments. The compounds in this EC range are not volatile (TPHCWG 1997c), so inhalation exposure to any of these PAHs as a result of contamination at hazardous waste sites is expected to be minimal under most circumstances. However, people may be exposed by inhaling dust or particles containing PAHs, or by inhaling PAHs released to the air, as vapors or aerosols, from shower water as a result of contamination of groundwater at hazardous waste sites.

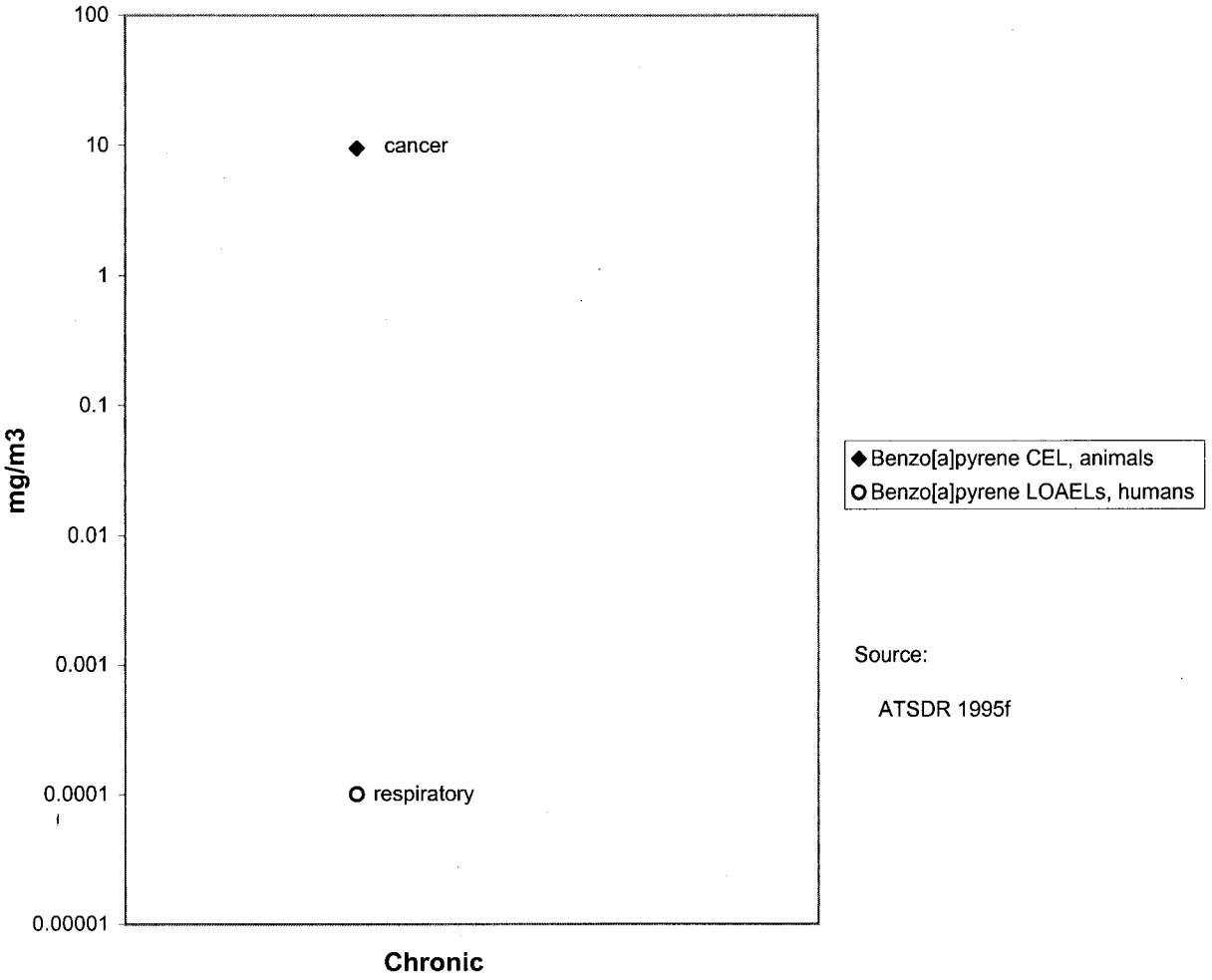
The few available inhalation LOAEL values for the combined aromatic E_{16}-EC_{35} fraction are summarized in Figure 6-10. More detailed information is available in the ATSDR (199%) toxicological profile.

6.2.3.2 Oral Exposure

Data for oral exposure, while more extensive than for inhalation exposure, are nonetheless limited. Hepatic effects appear to be a common sensitive end point of oral exposure to the PAHs in this combined fraction. Renal effects have been seen with some (ATSDR 1995f; EPA 1998b). Aplastic anemia and immunological/lymphoreticular effects have been seen at higher exposure levels.

Intermediate oral MRLs are available for two of the compounds in the $EC_{>16}$–EC_{21} fraction, fluorene and anthracene, based on subchronic studies in mice. The MRL for fluorene was based on hepatic effects (increased liver weight); the MRL for anthracene was based on the absence of any effects, including hepatic, in a similar study (ATSDR 1995f). An EPA-sponsored subchronic oral study of pyrene in mice was used by that agency as the basis for developing subchronic and chronic RfDs (EPA 1997a, 1998b). The critical effect was renal (nephropathy). Hepatic effects were not seen in this study, which is the only subchronic or chronic oral toxicity study of pyrene encountered. All four of the PAHs in this fraction that have been assessed for carcinogenicity by EPA have been classified in EPA Weight-of-Evidence Group D (not classifiable as to human carcinogenicity) (ATSDR 199%).

Figure 6-10. Aromatic $EC_{>16}$-EC_{35} Exposures Associated with Heath Effects - Inhalation



Source:

ATSDR 1995f

The only oral MRL available for compounds in the $EC_{>21}$- EC_{35} fraction is an intermediate MRL for fluoranthene, based on hepatic effects in mice. The sensitive noncancer effect of oral exposure to benzo(a)pyrene is developmental, also determined in animals.

Studies of the compounds in this fraction have focused primarily on potential carcinogenicity. Of the nine compounds in this EC range that have been assessed for carcinogenicity by EPA, seven have been classified in Group B2 (probable human carcinogen), and the remaining two, fluoranthene and benzo(g,h,i)perylene, in group D (ATSDR 1995f; EPA 1997a, 1998b). The evidence has come in large part from parenteral and dermal studies. Oral studies of carcinogenicity have been conducted for six of the PAHs in this EC fraction, with positive results for benzo(a)pyrene, benz(a)anthracene, and dibenz(a,h)anthracene, and with negative results for anthracene, fluoranthene, and fluorene (ATSDR 1995f).

The lowest reliable LOAEL values and the available MRLs for the combined aromatic $EC_{>16}$-EC_{35} combined fraction are summarized in Figure 6- 11, as are cancer risk levels. Information on pyrene is discussed above in this section. Additional information from EPA sources has been added for pyrene. More detailed information on the constituents of this fraction is available in the ATSDR (1995f) toxicological profile.

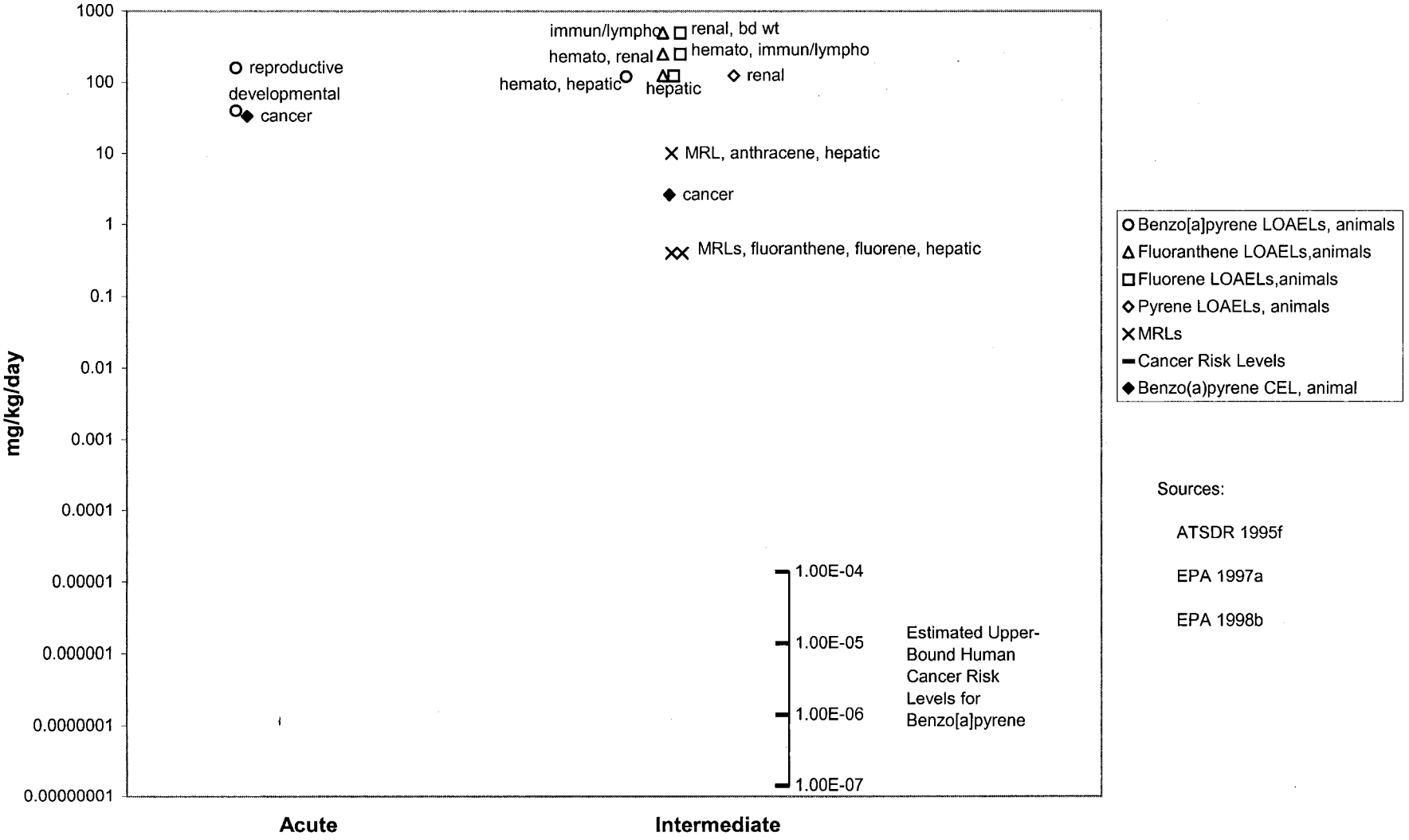
6.2.3.3 Dermal Exposure

The PAHs tend to be irritating to the skin. In addition, benzo(a)pyrene has been shown to cause immunological/lymphoreticular effects evidence as contact hypersensitivity or suppression of this response to other sensitizers. The PAHs classified as B2 carcinogens induce skin tumors following intermediate dermal application to animals (ATSDR 1995f).

6.2.4 Aliphatic EC_5-EC_8 Combined Fractions

EC_5-EC_6 Fraction: includes *n*-pentane, *n*-hexane, the dimethylbutanes and methylpentanes, cyclopentane, and some alkenes. *n*-Hexane is the only compound in this group that is the subject of an ATSDR toxicological profile; some information on commercial hexane (*n*-hexane plus branched and cyclic C_6 alkanes) is included in the same toxicological profile (ATSDR 1999b).

Figure 6-11. Aromatic $EC_{>16}$-EC_{35} Exposures Associated with Health Effects - Oral



Sources:

ATSDR 1995f

EPA 1997a

EPA 1998b

$EC_{>6}$-EC_8 Fraction: includes *n*-heptane, *n*-octane, some branched chain C_6-C_9 alkanes including the trimethylpentanes (note that other branched chain C_9 alkanes fall in the $EC_{>8}$ category) and cycloalkanes, including cyclohexane, methylcyclopentane, and methylcyclohexane, as well as some alkenes. None of these is the subject of an ATSDR toxicological profile.

6.2.4.1 Inhalation Exposure

Inhalation exposure for acute, intermediate or chronic durations to *n*-hexane causes peripheral neuropathy in humans and animals (ATSDR 1999b). The chronic MRL for *n*-hexane is based on this effect in humans. Respiratory and renal effects have been seen in animals exposed to *n*-hexane by inhalation at higher exposure levels than associated with peripheral neuropathy in the same studies. Calculation of human equivalent concentrations (HECs) using EPA dosimetric methodology, however, indicates that respiratory effects were seen in mice exposed subchronically to *n*-hexane at a HEC similar to that for neurological effects in the human study used as the basis for the chronic MRL (EPA 1998b). Thus, respiratory effects also may be sensitive, although confirmation of this in human studies is not available. The other compounds in the EC_5-EC_6 fraction do not appear to cause peripheral neuropathy (ATSDR 1999b; TPHCWG 1997c). Depression of the central nervous system has been seen at relatively high levels of exposure to *n*-hexane. *n*-Hexane has been classified as in weight-of-evidence Group D (not classifiable as to human carcinogenicity) (EPA 1989a).

Commercial hexane, which consists of a mixture of C_6 aliphatic compounds including 20-80% *n*-hexane and other straight, branched, and cyclic alkanes in the range of $EC_{5.68}$-$EC_{6.59}$,has been the subject of extensive recent testing as part of a EPA Test Rule under TSCA Section 4. Commercial hexane mixtures have the potential to represent the toxicity of the EC_5-EC_8 combined fraction better than any single compound. The non *n*-hexane components of commercial hexane, when tested separately as a mixture, do not cause peripheral neuropathy, whereas the commercial mixture containing *n*-hexane has been demonstrated to cause peripheral neuropathy in one study in rats (ATSDR 1999b; IRDC 1981). The commercial hexane mixtures tested under the Test Rule contained 53% *n*-hexane, 16% 3-methylpentane, 14% methylcyclopentane, 12% 2-methylpentane, 3% cyclohexane, 1% 2,3-dimethylbutane, and <1% other constituents. According to the TPHCWG (1997c), which developed an RfC for commercial hexane based on preliminary reports of these unpublished studies, the critical effects were respiratory (mucosal irritation in nasal turbinates and

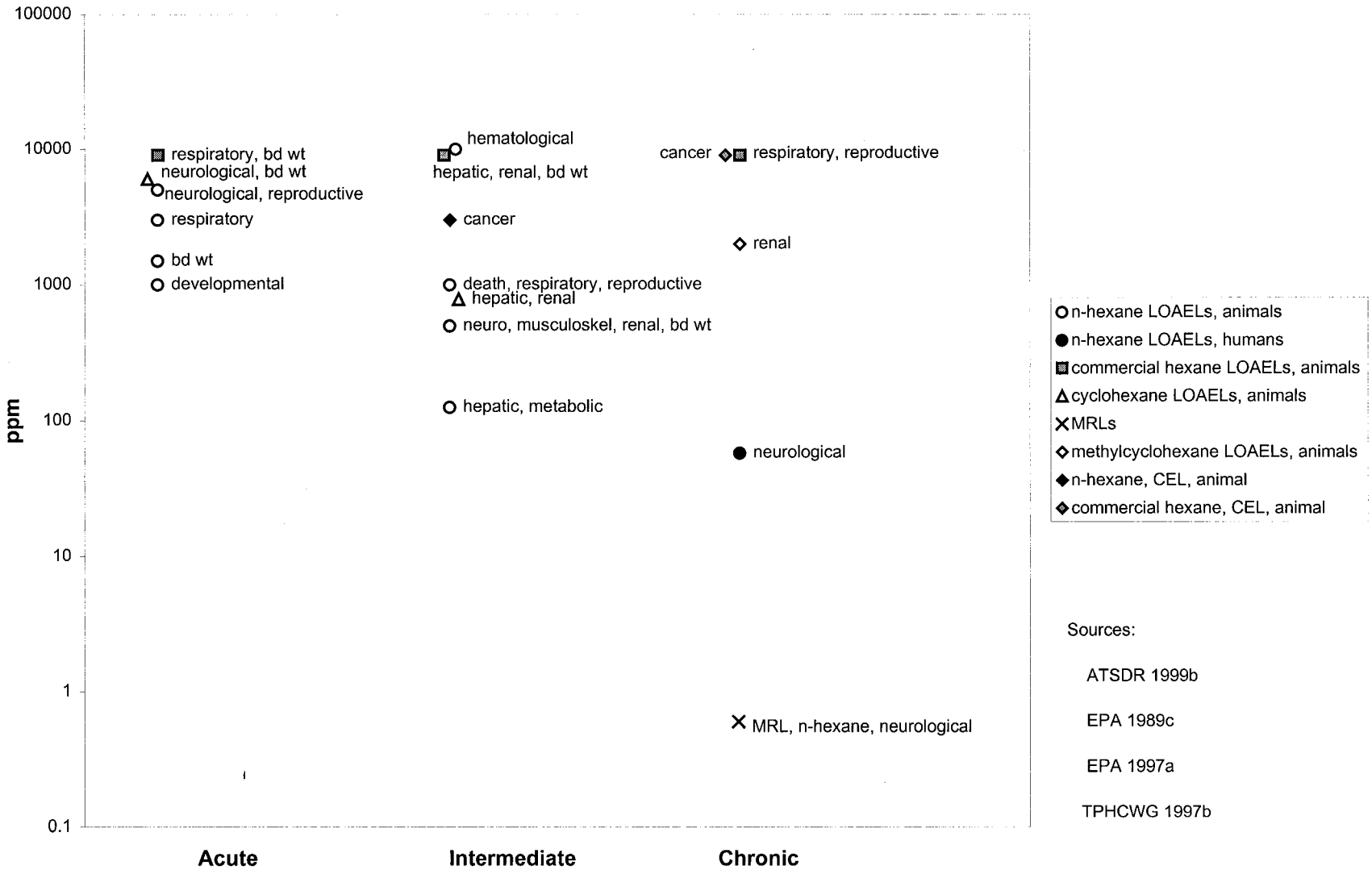
larynx in rats) and reproductive (decreased severity and incidence of cystic uterine endometrial hyperplasia in mice) in chronic studies. In addition, liver tumors developed in the female mice, indicating carcinogenic potential.

Cyclohexane also has undergone testing under EPA TSCA Section 4. The TPHCWG (1997c) summarized the preliminary report of the developmental toxicity study in rats, which indicates neurological effects (reduced response to a sound stimulus) in the dams exposed to cyclohexane by inhalation. Hepatic and renal effects were seen in published subchronic studies in animals. No histopathological changes in the peripheral nervous system were seen in a chronic study in animals (TPHCWG 1997c).

Two additional chemicals in the $E_{>6}$-EC_8 fraction that have been the subject of limited toxicity testing are *n*-heptane and methylcyclohexane. Both appear to cause depression of the central nervous system following relatively high inhalation exposures (EPA 1989b, 1989c). *n*-Heptane was suspected to have the potential to cause peripheral neuropathy because of its structural similarity to *n*-hexane and because it is metabolized, although to a much lesser extent, to the same type of metabolite (a γ-diketone) as is thought to mediate the neurotoxicity of *n*-hexane. The available human occupational and animal experimental studies, however, give no clear evidence that *n*-heptane causes peripheral neuropathy (EPA 1989b). Methylcyclohexane caused renal effects (medullary mineralization and papillary hyperplasia) in male but not in female rats or in other species exposed for 1 year by inhalation followed by an observation period; this study is the basis for an RfC derived by EPA (1997a). The renal effect appears to be associated with $\alpha_{2\mu}$-globulin nephropathy and, therefore, may be of questionable significance to human health. Both these compounds have been classified in Group D (not classified as to human carcinogenicity) (EPA 1989c, 1998b).

The lowest reliable LOAEL values for *n*-hexane are summarized in Figure 6-12, along with the available MRL. Because so few of the compounds in this fraction have been assessed by ATSDR, limited additional information from EPA sources and the TPHCWG (1997c) regarding commercial hexane, cyclohexane, and methylcyclohexane has been added. More detailed information is available in ATSDR (1997c) and the EPA and TPHCWG sources noted above.

Figure 6-12. Aliphatic EC_5-EC_8 Exposures Associated with Health Effects - Inhalation



Sources:

ATSDR 1999b

EPA 1989c

EPA 1997a

TPHCWG 1997b

6.2.4.2 Oral Exposure

Oral health effects information for the EC_5-EC_6 fraction is limited and is available mainly for *n*-hexane. *n*-Hexane caused peripheral neuropathy in rats given the compound subchronically and in chickens given the compound acutely and subchronically. The chicken is considered to be a valuable model for human neurotoxicity of this type. 2-Methylpentane and methylcyclopentane affected nerve conduction velocity in a subchronic study in rats, but were not as effective as *n*-hexane in that same study. Reproductive (testicular) and developmental effects have been seen in animals at higher doses of *n*-hexane than associated with neurological effects. No oral MRLs were derived for *n*-hexane because of the incompleteness of the database (ATSDR 1999b). *n*-Hexane has been classified as a Group D agent (not classifiable as to human carcinogenicity) (EPA 1989a).

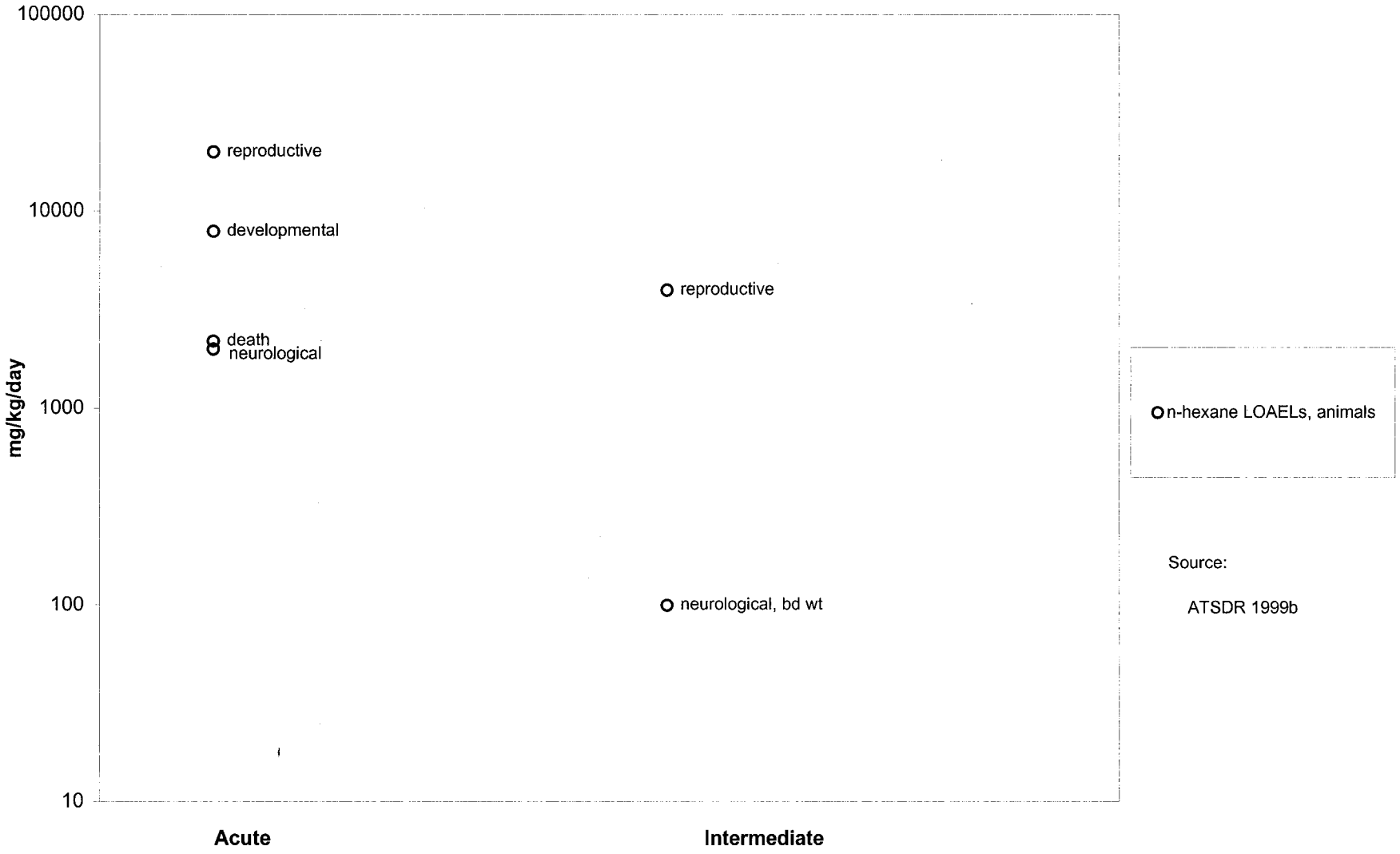
An oral 90-120-day study in rats of a commercial hexane containing 40% *n*-hexane, 24% each of 3-methylpentane and dimethylbutane, 9% cyclopentane, 2.5% cyclohexane, and 12% 2-methylpentane was conducted in comparison with *n*-hexane. This mixture includes compounds in both the EC_5-EC_6 and $EC_{>5}$-EC_8 range. Peripheral neuropathy was not seen when commercial hexane was tested at the same dose as was effective for pure *n*-hexane (ATSDR 1999b), but the dose of *n*-hexane resulting from this dose of commercial mixture was only 40% the effective dose of the pure *n*-hexane. Some evidence of carcinogenic potential has been reported in chronic inhalation studies in mice, as discussed in the previous section.

The lowest reliable LOAELs for *n*-hexane are plotted in Figure 6- 13. More detailed information, including some information on oral toxicity of related isomers and commercial hexane, is available in ATSDR (1997c).

6.2.4.3 Dermal Exposure

Some of the compounds in the combined $EC_{>9}$-EC_{16} fraction are known to be irritating to the skin and eyes, but little information is available to suggest systemic toxicity from dermal exposure.

Figure 6-13. Aliphatic EC_5-EC_8 Exposures Associated with Health Effects - Oral



6.2.5 Aliphatic $EC_{>8}$- EC_{16} Combined Fractions

$EC_{>8}$-EC_{10} fraction: includes *n*-nonane, *n*-decane, branched-chain C_9 -C_{10}, compounds, a few substituted cycloalkanes, and a few alkenes

$EC_{>10}$- EC_{12} fraction: includes *n*-undecane, *n*-dodecane, and pentylcyclopentane

$EC_{>12}$- EC_{16}fraction: *n*-tri-, tetra-, penta-, and hexadecane (Note that EC values for a number of branched and cyclic alkanes that potentially belong in these fractions were not listed by the TPHCWG [1997c1; see Appendix D: Table D-l for listing).

None of the individual compounds in the combined aliphatic $EC_{>8}$-EC_{16} fraction is the subject of an ATSDR toxicological profile. Some petroleum products, however, are mixtures primarily of aliphatic hydrocarbons in the range covered by this fraction. The TPHCWG (1997c) identifies JP-8 jet fuel as a mixture containing aliphatic petroleum hydrocarbons ranging from C_9-C_{16} and ATSDR has developed a toxicological profile on JP-8 (ATSDR 1998b). JP-8 contains up to 20% aromatics (C_{10}-C_{11}, $EC_{10.5}$-$EC_{12.99}$) (ATSDR 1998b; TPHCWG 1997b). Other petroleum products that are composed primarily of C_9 -C_{16}, aliphatics are JP-5, JP-7, and kerosene (fuel oil #l). These fuels also are the subjects of ATSDR toxicological profiles, and have at least one MRL (ATSDR 1995c, 1995g, 1998b). They contain approximately 16%, a maximum of 5%, and approximately 24% aromatic hydrocarbons, respectively. The jet fuels contain a number of additives such as antioxidants, metal deactivators, fuel system icing inhibitors, corrosion inhibitors, and static dissipaters. Stoddard solvent contains primarily C_9-C_{16}, aliphatics, with approximately 14% aromatics, and is also the subject of an ATSDR toxicological profile, but has no MRLs (ATSDR 1995b).

TPHCWG (1997c) also identifies a number of published and unpublished studies on dearomatized petroleum streams that correspond to portions of this range, and that contain at most 1.5% aromatics and more typically less than 0.1% aromatics. These studies on dearomatized petroleumstreams would appear to be a better basis for the assessment of health effects of this fraction, because they contain much smaller amounts of aromatics than do the petroleum products discussed in the previous paragraph and no additives. Their exact compositions and EC ranges were not reported, but EC numbers for the aliphatics tend to be close to the actual carbon numbers.

6.2.5.1 Inhalation Exposure

Hepatic effects are the most sensitive end points for inhalation exposure to JP-5, JP-7, JP-8, and kerosene (ATSDR 1995c, 199.58, 1998b). The available intermediate and chronic MRLs for these fuels are based on hepatic effects in animals. Neurological effects, particularly central nervous depression, have been seen in humans exposed acutely to JP-5 vapors, but exposure-effect relationships have not been established. Male rat $\alpha_{2\mu}$-globulin nephropathy occurred with exposure to JP-5 and JP-7, but this effect is not considered relevant to humans. A l-year exposure to JP-7 produced a small increase in the incidence of C-cell adenomas and kidney adenomas in male rats exposed to the vapor; the kidney adenomas may have been related to male rat $\alpha_{2\mu}$-globulin nephropathy, an effect with questionable relevance to human health.

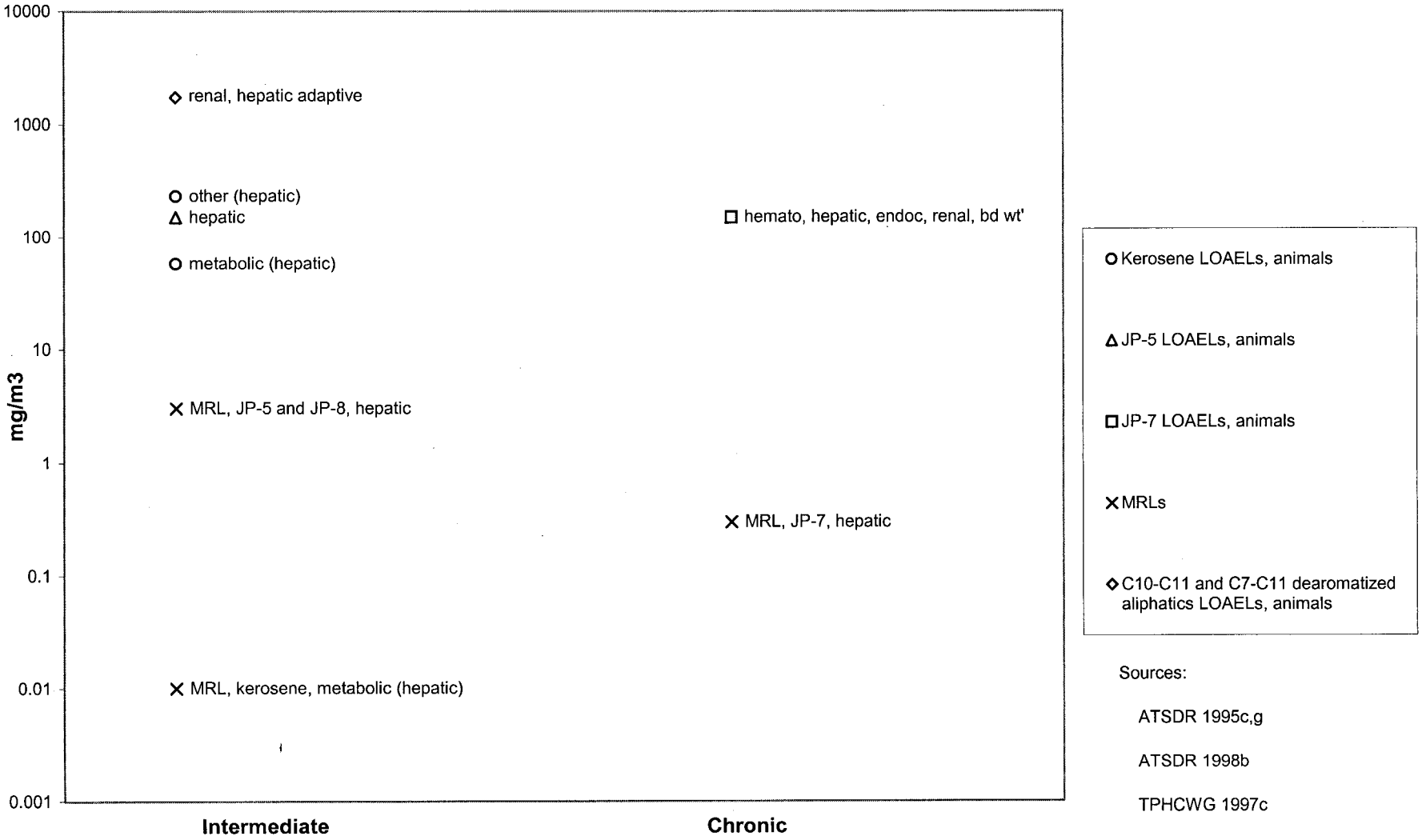
The inhalation studies of dearomatized petroleum streams included a C_{10}-C_{11} isoparaffinic solvent (branched chain alkanes), and C_7-C_{11} dearomatized white spirit (branched, straight and cyclic alkanes). Subchronic toxicity studies of these streams reported male rat nephropathy of the type that is of questionable relevance to humans health, according to the TPHCWG (1997c). In addition, increased liver weights were observed in male rats, but were said to be not significant. Developmental toxicity studies of these streams in rats revealed no developmental or maternal toxicity at the same exposure levels. These unpublished studies have been used as the basis for RfCs by the TPHCWG (1997c).

The lowest reliable LOAEL values for the jet fuels and kerosene discussed in this section are summarized in Figure 6-14, along with the available MRLs. Because these products have a significant aromatic component, limited additional information from the TPHCWG (1997c) regarding dearomatized petroleum streams has been added. More detailed information is available in the ATSDR toxicological profiles and the TPHCWG source noted above.

6.2.5.2 Oral Exposure

Oral data regarding JP-5, JP-7, JP-8, and kerosene were limited and judged inadequate for MRL development (ATSDR 19958, 1995c, 1998b). Hepatic effects and neurological effects have been seen from acute-duration oral exposure, but dose-effect relationships are either not well defined, or effects occurred at doses that also were fatal. Male rat nephropathy and decreased body weight were seen in

Figure 6-14. Aliphatic $EC_{>8}$-EC_{16} Exposures Associated with Health Effects - Inhalation



Sources:

ATSDR 1995c,g

ATSDR 1998b

TPHCWG 1997c

a 90-day oral study of JP-8 in male rats (Mattie et al. 1995) that was used by the TPHCWG (1997c) as the basis for an RfD, but ATSDR declined to derive an intermediate oral MRL because of the general lack of data and limitations of this study.

Subchronic studies of the dearomatized petroleum streams in rats were conducted on C_9-C_{12}, and C_{10}-C_{13} dearomatized aliphatic mixtures containing branched, straight, and cyclic alkanes, and a C_{11}-C_{17} isoparaffinic solvent containing branched and cyclic alkanes. Two of these studies reported male rat nephropathy. All three studies reported hepatic effects including hepatocellular hypertrophy and increased liver weight. Developmental toxicity was not seen at the same doses in a study of a similar mixture in rats. These unpublished subchronic studies were used as the basis for RfDs by the TPHCWG (1997c).

The lowest reliable LOAEL values for the jet fuels and kerosene discussed in this section are summarized in Figure 6-15. Because these products have a significant aromatic component, limited additional information from the TPHCWG (1997c) regarding dearomatized petroleum streams has been added. More detailed information is available in the ATSDR toxicological profiles, the TPHCWG source noted above, and Section 6.3.

6.2.5.3 Dermal Exposure

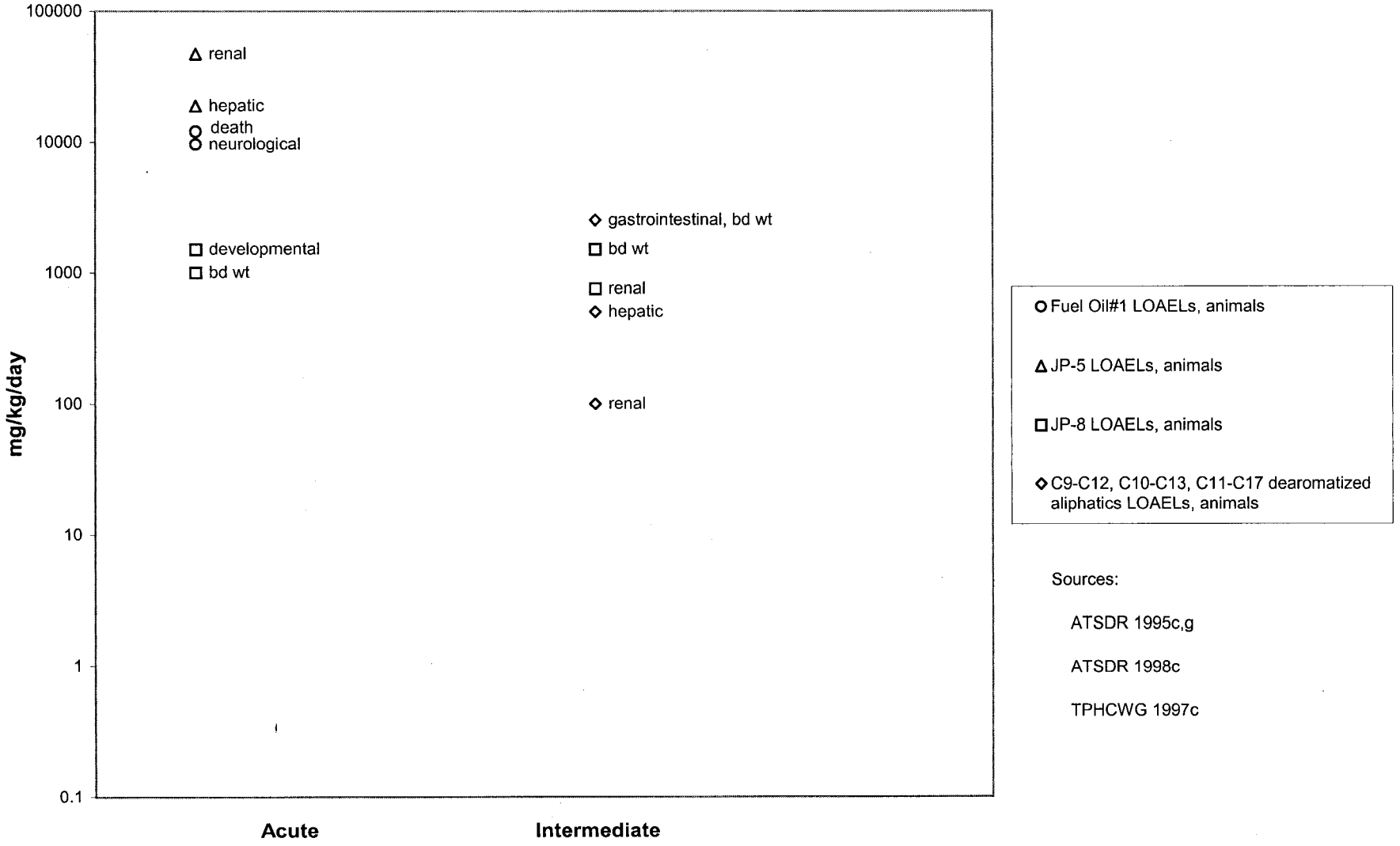
Information on the health effects of dermal exposure to JP-5, JP-7, and JP-8, and kerosene is limited. Skin and eye irritation are well documented, but effects from systemic absorption are not (ATSDR 1995c**,** 19958, 1998b).

6.2.6 Aliphatic $EC_{>16}$-EC_{35} Combined Fractions

$EC_{>16}$-EC_{21} fraction: includes *n*-hepta-, *n*-octa-, and *n*-nonadecane; and *n*-eicosadecane

$EC_{>21}$-EC_{35} fraction: includes *n*-heneicosane, *n*-docosane, *n*-tetracosane, and *n*-hexacosane. (Note that aliphatic compounds other than the above straight-chain alkanes were not listed by the TPHCWG [1997b] as constituents of petroleum and petroleum-based fuels that are the focus of the fraction-selection approach. See Appendix D, Table D-l.) Petroleum products such as mineral-based crankcase oil and mineral-based hydraulic fluids, however, contain branched and cyclic

Figure 6-15. Aliphatic $EC_{>8}$-EC_{16} Exposures Associated with Health Effects - Oral



Sources:

ATSDR 1995c,g

ATSDR 1998c

TPHCWG 1997c

aliphatics within these equivalent carbon ranges, as do food-grade and medicinal-grade mineral oils. Although ATSDR toxicological profiles are available for mineral-based used crankcase oil and mineral-based hydraulic fluids (ATSDR 1997b, 1997c), these products contain additives and contaminants, including substantial levels of aromatics and metals (used crankcase oil) and organophosphate esters (hydraulic fluids). Little information is available regarding health effects of these products. No MRLs have been derived. The TPHCWG (1997c) has reviewed data regarding food and medicinal grade mineral oils, which are relatively pure and therefore a better choice to represent this fraction.

6.2.6.1 Inhalation Exposure

No information was located on the potential health effects of inhalation exposure to compounds or mixtures of petroleum hydrocarbons that fall within this fraction.

6.2.6.2 Oral Exposure

Purified mineral oils have been used medicinally and in foods. Subchronic toxicity studies of selected mixtures of mineral oil hydrocarbons (composed primarily of branched chain alkanes or cyclic alkanes) in F344 rats have identified the liver and the mesenteric lymph nodes as potential targets of toxicity for these mineral oils. The TPHCWG (1997c) derived chronic RfDs for low and high molecular weight mineral oils based on the hepatic effects (lipid granulomas) seen in these studies. The effect on the mesenteric lymph nodes (histiocytosis), which occurred at lower exposure levels than did the hepatic effects, was judged a nonadverse, adaptive response to the ingestion of foreign material (TPHCWG 1997c). Subchronic oral toxicity testing has also been conducted with low- and intermediate-molecular weight paraffin waxes, which contain a high proportion of straight chain alkanes and also branched alkanes and small amounts of cyclic alkanes, with C ranges primarily within this fraction range (Smith et al. 1996). Results indicate that these mixtures have toxicity similar to that of the oils for which the RfDs were derived. Strains of rats other than F344 appeared to be less sensitive to these mixtures.

Hepatic lipid granulomas have also been seen in humans exposed to mineral oils through the diet and by ingestion of medicinal mineral oils, but doses associated with the effect in humans are not known. According to TPHCWG (1997c), the granulomas in humans were circumscribed lesions with no

inflammation, fibrosis, or significant liver dysfunction, whereas the granulomas in F344 rats were reactive with associated inflammation and occasional parenchymal cell necrosis.

The LOAELs identified for the “low” molecular weight mineral oils (C_{16}-C_{35}) are plotted in Figure 6-16. Additional information on health effects is provided in the review by TPHCWG (1997c).

6.2.6.3 Dermal Exposure

Information regarding health effects of dermal exposure to this fraction was not encountered in the cited source (TPHCWG 1997c).

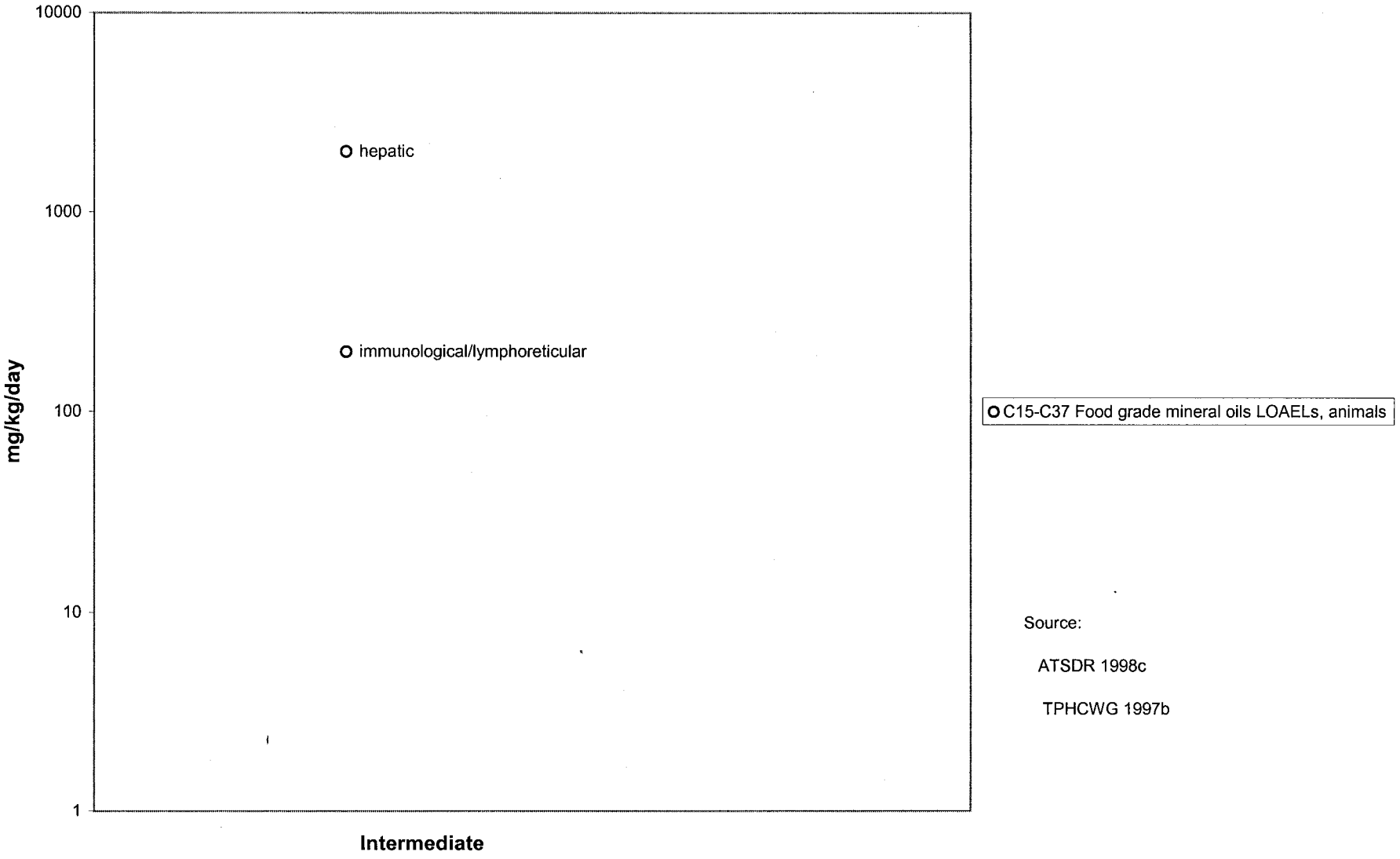
6.3 DISCUSSION OF HEALTH EFFECTS FOR WHOLE PETROLEUM PRODUCTS

Whole petroleum products are generally complex mixtures of hydrocarbons of varying carbon number and additives (usually representing a smaller weight percentage of the whole mixture) of varying chemical identities that are added to impart special qualities or enhance particular functional properties of the whole petroleum product. Additional impurities may be generated during use of the product. Non-hydrocarbon additives and impurities are not included in the definition of TPH. Toxicological information on important petroleum products that are the subjects of other ATSDR toxicological profiles, and on other petroleum products that are the subject of assessment by other agencies, is briefly reviewed in this section. Such information may be useful in characterizing acute exposure to fresh spills of petroleum products, but its usefulness is limited because of the limited availability of MRLs, the variability in the composition of petroleum products, and the change in composition due to environmental fate and transport processes. The whole petroleum products that have compositions similar to the transport fractions have been discussed in Section 6.2.

6.3.1 Jet Fuels

Jet fuels are middle distillates of petroleum crude oils that are composed of hydrocarbons generally coming off distillation columns at temperatures between 150 and 300 ºC (ATSDR 1998b; IARC

Figure 6-16. Aliphatic $EC_{>16}$-EC_{35} Exposure Levels Associated with Health Effects - Oral



1989c). Kerosene-type jet fuels such as JP-5, JP-7, and JP-8 have the same basic composition as kerosene (consisting predominately of hydrocarbons with carbon numbers in the range of C_9-C_{16}), whereas "wide-cut" jet fuels such as JP-4 are blends of kerosene and lower-boiling naphtha streams (C_4-C_{16}). Jet fuels are refined under more stringent conditions than kerosene and contain various additives (anti-oxidants, dispersants and/or corrosion inhibitors) not found in kerosene. The exact chemical composition varies depending on the source of crude oil and additives included in the formulated product. Generally, aliphatic hydrocarbons represent the major part and aromatic hydrocarbons represent about 10-20% of kerosene and jet fuels. The benzene content of kerosenetype jet fuels is generally <0.02%, whereas "wide-cut" jet fuels typically contain more benzene (normally <0.5%). PAHs, with boiling points above 300 ºC, are generally excluded from jet fuels and kerosene.

Health effects of concern from exposure to jet fuels include eye and skin irritation from acute direct contact; respiratory, neurotoxic and gastrointestinal effects from acute accidental ingestion; and possible hepatic damage from inhalation exposure of intermediate duration as indicated by results from animal studies (ATSDR 1998b).

ATSDR (1998b) derived an intermediate-duration inhalation MRL of 3 mg/m^3 for jet fuels JP-5 and JP-8, based on a LOAEL for hepatocellular fatty changes and vacuolization in mice exposed continuously for 90 days to vapors of JP-5 at a concentration of 150 mg/m^3 (Gaworski et al. 1984). The exposure concentration was converted to a human equivalent exposure concentration (853 mg/m^3) by multiplying by the ratio of the alveolar ventilation rate divided by the body weight of mice to the same parameters for humans. The human equivalent concentration was divided by an uncertainty factor of 300 (10 for interspecies variability, 3 for intraspecies variability, and 10 for the use of a LOAEL) to derive the MRL.

ATSDR (1998b) derived no other MRLs for JP-5 or JP-8 (e.g., for acute or chronic inhalation exposures, or for oral exposures of any duration), due to the lack of data suitable for MRL derivation.

ATSDR (1995c) derived an intermediate-duration inhalation MRL of 9 mg/m^3 for JP-4 based on a LOAEL of 500 mg/m^3 for hepatic fatty degeneration in mice exposed continuously to the vapor for

90 days. The MRL was derived from this LOAEL by dosimetrically adjusting to a human equivalent concentration and applying an uncertainty factor of 300 (10 for the use of a LOAEL, 3 for interspecies extrapolation, and 10 for human variability). ATSDR (199%) derived a chronic-duration inhalation MRL of 0.3 mg/m^3 for JP-7, based on a LOAEL of 150 mg/m^3 for hepatic inflammation in rats exposed to the vapor (6 hours/day, 5 days/week) for 1 year and observed for an additional year. The MRL was calculated from this LOAEL by dosimetrically adjusting to a human equivalent continuous exposure concentration and applying an uncertainty factor of 300 (10 for the use of a LOAEL, 3 for interspecies extrapolation, and 10 for human variability).

ATSDR (199%) derived no other MRLs for jet fuels JP-4 and JP-7, due to the lack of additional suitable inhalation data and the absence of data for oral exposure to these jet fuels.

ATSDR (1995c) found no studies regarding cancer in humans exposed to the jet fuels JP-4 and JP-7. Inhalation animal studies provided no evidence that JP-7 was carcinogenic (Air Force 1991). A l-year study of rats and mice exposed by inhalation to vapors of JP-4 was identified in which increased tumors were found in the respiratory tract of female rats and mice, increased renal tumors (associated with the $\alpha_{2\mu}$-globulin nephropathy syndrome) were found only in male rats, and increased liver tumors were found in female, but not male mice (Bruner et al. 1993). ATSDR (1995c) concluded that the animal data provided equivocal evidence for the carcinogenicity of JP-4 and that there was insufficient evidence to draw conclusions regarding the carcinogenic potential of JP-4 or JP-7 in humans.

ATSDR (1998b) concluded from a review of several studies of mice dermally exposed to jet fuels (including JP-5 and Jet A) that chronic dermal application of jet fuels can act as a skin carcinogen, but noted that further investigation is needed to more fully elucidate “the impact of dermal exposure of jet fuels on humans.”

IARC (1989d) concluded that there was inadequate evidence for the carcinogenicity of jet fuel in humans and animals, but noted that there is limited evidence for the carcinogenicity in experimental animals of straight-run kerosene and hydrotreated kerosene. IARC’s review included: a cohort mortality study that found no increased cancer risk in men exposed to jet fuel, aviation kerosene, and other fuels in the Swedish Air Force; elevated risk for kidney cancer in men exposed to jet fuel in a

Canadian case-control study; and both positive and negative findings for skin cancer in studies of mice dermally exposed to jet fuels.

6.3.2 Fuel Oils

Fuel oils refined from crude petroleum can be classified either as *distillate fuels* consisting predominately of distilled process streams or as *residual fuels* consisting of residues remaining after distillation or blends of residues and distillates (ATSDR 19958; IARC 1989b). Both types of fuel oils are complex mixtures of aliphatic hydrocarbons (representing approximately 80-90% of these oils) and aromatic hydrocarbons (representing 10-20%). Light distillate fuels (e.g., fuel oil #l, straight-run kerosene) consist primarily of hydrocarbons in the C_9-C_{16}, whereas hydrocarbons in middle distillate fuels (e.g., fuel oil #2) may range from approximately C_{11}-C_{20}. Diesel fuels are similar to fuel oils with the exception that the diesel fuels contain additives. Light and middle distillate fuels generally contain less than 5% polycyclic aromatic hydrocarbons. Heavier fuel oils (e.g., fuel oil #4 and marine diesel fuel) may contain up to 15% distillation residues and more than 5% polycyclic aromatic hydrocarbons. Residual fuel oils are more complex in composition than distillate fuels, and can contain significant portions of compounds with sulfur and nitrogen.

Reports of cases of accidental ingestion of kerosene identify respiratory effects (e.g., pulmonary edema and difficulty in breathing from aspiring the liquid into the lungs), nervous system depression, and gastrointestinal irritation as effects of concern from acute exposure to fuel oils (ATSDR 1995g). These effects (and others including skin and eye irritation, and increased blood pressure) have been observed in humans in a few cases after inhalation and/or dermal acute exposures. Animal studies provide supporting data for neurological impairment from acute inhalation exposure to fuel oil #2 and hepatic effects (including decreased blood glucose levels and hepatocellular fatty changes and vacuolization) from intermediate-duration exposure to fuel oil #l and jet fuel JP-5.

ATSDR (19958) derived an acute-duration inhalation MRL of 0.02 mg/m^3 for diesel fuel (fuel oil #2) based on observations of mild transient ataxia and disturbed gait in mice exposed for 8 hours/day for 5 days to vapors of diesel fuel #2 at concentrations as low as 65 mg/m^3 (Kainz and White 1984). The LOAEL was adjusted to a continuous exposure basis and divided by an uncertainty factor of 1,000 (10 for intraspecies variability, 10 for interspecies variability, and 10 for the use of a LOAEL). ATSDR (1995g) did not discuss the potential applicability of this MRL to other fuel oils.

ATSDR (19958) derived an intermediate-duration MRL of 0.01 mg/m^3 for kerosene (also called fuel oil #l) based on a LOAEL for decreased blood glucose levels (thought to be indicative of hepatic effects) in rats exposed 6 hours/day, 6 days/week for 14 weeks to fuel oil #l at concentrations of 58 mg/m^3 (Starek and Vojtisek 1986). The LOAEL was adjusted to a continuous exposure basis and divided by an uncertainty factor of 1,000 (10 for intraspecies variability, 10 for interspecies variability, and 10 for the use of a LOAEL). ATSDR (19958) did not discuss the potential applicability of this MRL to other fuel oils, but cited, as supporting data for the MRL, findings of hepatocellular changes and vacuolization in mice exposed continuously to 150 mg/m^3 IP-5 for 90 days, and findings of no systemic or neurological effects in rats or dogs exposed to a deodorized kerosene concentration of 100 mg/m^3, 6 hours/day, 5 days/week for 13 weeks.

ATSDR (1995g) did not derive chronic inhalation MRLs or any oral MRLs (for any duration of exposure) because suitable data were not available.

From a review of available human and animal studies, ATSDR (1995g) concluded that epidemiological studies have provided “only equivocal evidence of an association between cancer and exposures to fuel oils” and that animal studies suggest that dermal exposure to fuel oils can produce skin or liver cancer. ATSDR (1995g) noted that the animal studies are restricted to one species (mice) and not all studies found carcinogenic responses. The conclusion was drawn that “further investigation utilizing other species is required to more fully elucidate the mechanism of dermal carcinogenesis and the impact of dermal exposure of fuel oils on humans.”

Based on their review, IARC (1989b) concluded that there was inadequate evidence for the carcinogenicity in humans of fuel oils; sufficient evidence for the carcinogenicity in experimental animals of residual (heavy) fuel oils; limited evidence for the carcinogenicity in experimental animals of fuel oil #2; sufficient evidence for the carcinogenicity in experimental animals of light and heavy catalytically cracked distillates, of light and heavy vacuum distillates and of cracked residues, all derived from the refining of crude oil; and limited evidence for the carcinogenicity in experimental animals of straight-run kerosene. Overall evaluations were made that residual (heavy) fuel oils are possibly carcinogenic to humans (Group 2B), and that distillate (light) fuel oils are not classifiable as to their carcinogenicity to humans (Group 3).

6.3.3 Automotive Gasoline

Gasoline is a complex mixture of volatile petroleum-derived hydrocarbons, additives, and blending agents (ATSDR 1995a; IARC 1989a). The composition of gasoline varies widely depending on the composition of the crude oil from which it is refined, the refining processes used, the type and relative amount of different petroleum refining streams blended in the finished product, and the types and amounts of nonhydrocarbon compounds added to enhance or impart specific functional properties of the gasoline. Specific market conditions, partly in response to regulations, mandate the refining and manufacturing of certain gasolines. Gasoline contains predominately hydrocarbons in the C_4-C_{12} range, with the following typical distributions: alkanes (4-8 wt%); alkenes (2-5 wt%); isoalkanes (25-40 wt%); cycloalkanes (3-7 wt%); cycloalkenes (l-4 wt%); and total aromatics (20-50 wt%). The benzene content of gasoline is 0.12-3.5% (see Table E-l .b for additional detail regarding individual hydrocarbon constituents). Additives found in gasoline include anti-knock agents (e.g., tetraethyllead), lead scavengers (e.g., 1,2-dibromoethane), detergents, anti-rust agents (e.g., sulfonates), antioxidants (e.g., p-phenylenediamine), and anti-icing agents (e.g., alcohols). Leaded gasoline is no longer allowed to be used by on-road vehicles, though it still is used in farm machinery boats, competetive vehicles, and in piston engine airplanes. (EPA 1998d). A variety of products are added to gasoline to boost octane, including ethanol and MTBE.

Acute-duration inhalation, oral, or dermal exposures to gasoline have been associated with irritation at portals of entry in humans, and high-level inhalation or oral acute exposure produces symptoms of transient neurological impairment such as headache, nausea, dizziness, euphoria, and drowsiness (ATSDR 1995a). Acute ingestion of large amounts of gasoline also produces respiratory effects such as pneumonitis and pulmonary edema due to the aspiration of gasoline. Chronic exposure to gasoline vapors by intentional inhalation also has been associated with symptoms providing evidence for more permanent neurological damage in humans such as postural tremor, abnormal gait, and affected speech. The relative degrees to which hydrocarbons and additives such as lead contribute to gasoline-induced neurological impairment are unknown. Studies with rats and mice with chronic inhalation exposure to gasoline vapors have found hepatocellular tumors in female mice, and $\alpha_{2\mu}$-globulin nephropathy and related renal tumors in male rats. The renal tumors are believed to be unique to male rats and of questionable relevance to humans.

ATSDR (1995a) derived no inhalation or oral MRLs for gasoline, “because of the variability in the composition of gasoline;” the toxicity would depend on the specific composition. ATSDR (1995a)

also commented, regarding oral exposure, that there is no "quantitative information on adverse effects other than $\alpha_{2\mu}$-globulin nephropathy in male rats," an end point that is considered "not relevant to human risk assessment."

Numerous epidemiology studies have examined possible relationships between exposure to gasoline and development of various types of cancer in humans, but none of the studies were adequate to conclusively demonstrate that exposure to gasoline causes cancer in humans (ATSDR 1995a). The most common problems with these studies were the failure to adequately characterize exposure and the failure to control for confounding exposures to other fuels and exhaust emissions. In a chronic inhalation study, exposure to whole vapors of unleaded gasoline produced an increased incidence of renal tumors in male rats and liver tumors in female mice (MacFarland et al. 1984b). The renal tumors in male rats were considered to arise as a result of a process involving $\alpha_{2\mu}$-globulin accumulation, a process not expected to occur in humans. ATSDR (1995a) further questioned the relevance of the MacFarland findings, because the animals were exposed to whole vapors of gasoline and "gasoline emissions found in the environment contain lower concentrations of hydrocarbons with very low vapor pressures" than those found in whole vapors of gasoline.

EPA (1987c) classified gasoline as a Group B2 compound, a probable human carcinogen, based on inadequate evidence of carcinogenicity in humans and sufficient evidence in animals. This evaluation was made before EPA adopted a policy excluding $\alpha_{2\mu}$-globulin-related renal tumors in male rats from cancer weight-of-evidence classifications. EPA derived an inhalation unit risk of 2.1×10^{-3} ppm for gasoline based on an analysis of tumor incidence data for hepatocellular adenomas and carcinomas in female mice exposed to unleaded gasoline vapors for 2 years (MacFarland et al. 1984b). EPA has not published a more recent classification for gasoline.

IARC (1989a) concluded that there was inadequate evidence for carcinogenicity of gasoline in humans and limited evidence for carcinogenicity of unleaded automotive gasoline in experimental animals (the evidence in MacFarland et al. [1984a]). IARC (1989a) classified gasoline-as "possibly carcinogenic to humans (Group 2B)," based on the preceding conclusions and supporting data showing that gasoline induces unscheduled DNA synthesis in mice *in vivo* and in mouse, rat and human hepatocytes *in vitro;* that light, straight-run naphtha and light catalytically cracked naphtha petroleum refinery streams used to blend gasoline produce skin tumors in dermally exposed mice; ant that gasoline components such as benzene and 1,3-butadiene are known or suspected carcinogens.

6.3.4 Various Petroleum Refinery Streams

A number of health effects studies in animals of petroleum streams that correspond with the transport fractions have been reviewed by the TPHCWG (1997c); however, most of these are unpublished industry studies.

6.3.5 Stoddard Solvent

Stoddard solvent is a petroleum distillate mixture of C_7-C_{l2} hydrocarbons, approximately 80-90% aliphatics (30-50% linear and branched alkanes, and 30-40% cyclic alkanes) and l0-20% aromatics (not PAHs). It is similar to white spirits, which is also included in the toxicological profile on Stoddard solvent (ATSDR 1995b). For additional detail, see Section 3.2 and Table E-2.b. Data regarding the health effects of Stoddard solvent in either humans or animals are limited and were judged inadequate for MRL development. Upper respiratory irritant effects were seen in animals exposed by inhalation for acute and intermediate durations; these appear to be the most sensitive effects by the inhalation route. Male rat nephropathy has been reported in intermediate inhalation studies, but is not considered relevant to human health. No oral studies were located. Information on the potential carcinogenicity of Stoddard solvent is inadequate.

6.3.6 Mineral-Based Crankcase Oil

Mineral-based crankcase oil is a petroleum product that is a complex mixture of low and high molecular weight (C_{15}-C_{50}) aliphatic and aromatic hydrocarbons, metals, and additives. The chemical composition of mineral-based crankcase oil varies widely, depending on the original crude oil, the processes used in refining, the types of additives included in the oil, the efficiency and type of engine in which it is used, the type of fuel used in the engine, and the length of time the oil was used in an engine. The hydrocarbon constituents are mainly straight and branched chain alkanes, cycloalkanes, and aromatics (see Table E-5.b for additional detail). Additives (which can account for-up to 20% of the weight of oil formulations) include detergents, metallic salts (e.g., molybdenum and zinc salts), and organometallic compounds. Metals (e.g., cadmium, lead and zinc) and PAHs have been demonstrated to increase in oil with continued use in an engine.

Studies examining petroleum-stream stocks used to formulate mineral-based crankcase oil indicate that these stocks are nontoxic relative to used crankcase oils; therefore, the toxicity of used oils has been attributed to additives present in the oil or to decomposition products or contaminants that accumulate in the oil with use (ATSDR 1997c). Studies of mechanics and auto-workers exposed to used mineral-based crankcase oil found elevated incidence of skin rashes, anemia, headaches and tremors, but these studies do not establish a causal relationship with exposure to used crankcase oil, due to several limitations of the studies including the likelihood that the workers were exposed to other chemicals which may have caused the effects. There are only a few toxicological studies of animals exposed to mineral-based crankcase oil. Acute exposures to mists of used mineral-based crankcase oil were irritating to the eyes and upper respiratory tract of some volunteer human subjects. Studies of rats ingesting large single doses (9,000-22,500 mg/kg) of used mineral-based crankcase oil found no adverse health effects other than diarrhea. Cattle that ingested an unknown amount of used mineral-based crankcase oil while grazing in contaminated pastures exhibited several health effects including death, anemia, and neurological dysfunction; it was postulated that the observed effects were caused by metals (molybdenum and lead) in the oil. Long-term dermal application of used mineral-based crankcase oil to the skin of mice produced an increased incidence of dermal papillomas and carcinomas and increased levels of DNA adducts associated with reactive metabolites of PAHs. The carcinogenicity of used mineral-based crankcase oil has been correlated with the PAH content of oils. ATSDR (1997e) judged that no meaningful MRL values could be derived for used mineral-based crankcase oil, due to the limitations of the toxicological data on used mineral-based crankcase oils and the wide compositional variance among used mineral-based crankcase oils.

EPA (1998b) and IARC (1996) have not classified used mineral-based crankcase oil as to its carcinogenicity in humans. IARC (1984, 1987) noted that exposure to mineral oils used in a variety of occupations (including mulespinning, metal machining, and jute processing) has been strongly and consistently associated with increased occurrence of squamous-cell cancers of the skin, especially of the scrotum, but that production processes have changed over time so that more modern, highly refined oils contain smaller amounts of “contaminants, such as polycyclic aromatic hydrocarbons.” IARC (1987) judged that there was sufficient evidence for the carcinogenicity of untreated and mildly-treated mineral oils in humans and animals, whereas there was inadequate evidence for the carcinogenicity of highly-refined mineral oils in humans or animals.

6.3.7 Mineral Oil Hydraulic Fluids

Most mineral oil hydraulic fluids are made from processed petroleum crude oils that are blended with various types of nonhydrocarbon additives to impart specific, use-related properties to the fluid (ATSDR 1997b). The carbon number range of hydrocarbons in hydraulic fluids varies depending on the intended application of the fluid, but mostly is in the range of C_{15}-C_{50}. Toxicity data for mineral oil hydraulic fluids are restricted to acute lethality studies of rats exposed by gavage or by inhalation to several types of mineral oil hydraulic fluids, and single-dose gavage neurotoxicity tests that found no effects in chickens.

ATSDR (1997b) did not derive inhalation or oral MRLs for mineral oil hydraulic fluids for any duration of exposure, because of the lack of suitable data.

IARC (1984) reviewed the evidence that certain types of mineral oils are carcinogenic in animals, whereas other types are not. IARC (1984) concluded that mineral oil is not classifiable as to its carcinogenicity, because of the apparent dependence of mineral oil's carcinogenic activity in animals on the chemical makeup of the crude oil starting material, the presence of additives and the conditions of use.

6.3.8 Asphalt

Asphalts are complex mixtures containing relatively high molecular weight hydrocarbons, predominantly cyclic alkanes and aromatic compounds (IARC 1985). They also contain some sulfur-, nitrogen- and oxygen-containing compounds and heavy metals. They are viscous liquids or solids. Inhalation studies of these mixtures in animals have involved heating the materials to produce fumes, which is relevant to human occupational exposure (e.g., roofing, road surfacing), but not particularly relevant to exposure resulting from contamination at hazardous waste sites. Respiratory effects were seen in these studies. Respiratory effects were reported in workers who were exposed to fumes of asphalts. No oral studies were reported. IARC concluded that there is sufficient evidence that extracts of asphalts (applied to the skin of experimental animals in solvents such as benzene or toluene or injected subcutaneously) are carcinogenic to animals. Evidence for undiluted asphalts ranged from limited to inadequate, depending on type of asphalt. IARC (1985) concluded that there is inadequate evidence that asphalts alone are carcinogenic to humans

6.3.9 Crude Oil

ATSDR has not prepared a toxicological profile on crude oil. IARC (1989c) prepared a monograph on crude oils, from which the following information is summarized. Crude oils are exceedingly complex mixtures that vary greatly depending on their source. The bulk of chemicals in crude oils are hydrocarbons: straight, branched and cyclic alkanes; and aromatics including benzene, alkylbenzenes, naphthalenes and PAHs. Non-hydrocarbon constituents of crude oil include sulfur-, nitrogen-, oxygen-and metal-containing compounds.

No studies of potential health effects from inhalation exposure were located. Acute oral administration of crude oil to animals has resulted in hepatic effects and development effects. Aspiration of crude oil by a laborer resulted in pneumonia and hepatic and renal effects. Petroleum field workers who had direct dermal contact with crude oil developed adverse dermal effects, including dryness and hyperkeratosis.

A number of studies of the carcinogenicity of dermal application of crude oil to animals have been reviewed by IARC (1989c), which concluded that there is limited evidence for the carcinogenicity of crude oil to experimental animals. A cohort study of U.S. petroleum-producing and pipeline workers, and case control studies that included exposure during crude oil exploration and production, were evaluated by IARC (1989c), which concluded that there is inadequate evidence for the carcinogenicity of crude oil in humans.

An additional monograph on occupational exposures in petroleum refining (IARC 1989e) concluded that there is limited evidence that working in petroleum refineries entails a risk of skin cancer and leukemia. Exposures during refining, however, are not particularly relevant to exposures resulting from contamination of hazardous waste sites with crude oil.

6.4 TOXICOKINETICS

Overview. Because TPH is a broadly defined entity consisting of complex mixtures of hydrocarbons of varying chemical composition (due to differences in original petroleum products and differential, time-dependent, fate and transport of components within any particular TPH mixture), this section discusses available information for absorption, distribution, metabolism and excretion of components and petroleum products corresponding to the transport fractions of TPH. Limited additional information regarding the

more heterogenous whole petroleum products can be found in the ATSDR toxicological profiles and other assessments of these products referenced in Section 6.3. In general, however, there is little information regarding toxicokinetics of these heterogeneous products and the discussions often deal with the individual constituents, including additives and impurities that are not petroleum hydrocarbons, and hydrocarbon mixtures that are similar to portions of the product.

Hydrocarbons in the aromatic $EC_{>9}$ –EC_{16}, fraction may be readily absorbed following inhalation or oral exposure, based on studies with humans and animals exposed to the BTEXs. BTEXs are absorbed by the skin to a lesser extent, especially with exposure to vapors. BTEXs and their metabolites are widely distributed throughout tissues and organs following absorption. BTEXs are metabolized (via oxidative metabolic pathways involving cytochrome P-450 oxidases and conjugation reactions with glucuronides, sulfates, glutathione, or amino acids) to more water-soluble metabolites that are excreted predominately in urine. Metabolism represents a toxification pathway for some effects of certain BTEXs (e.g., cancer and hematopoietic effects appear to be caused by reactive metabolic intermediates of benzene) and a detoxification pathway for other effects (e.g., neurological effects from acute exposure to toluene). In addition to urinary excretion of metabolites, BTEXs are eliminated by exhalation of unchanged parent compound and fecal excretion (ATSDR 1994, 1995d, 1997a, 1999a).

Hydrocarbons in the aromatic $EC_{>9}$ –EC_{16} fraction may be absorbed following inhalation, oral, or dermal exposure, based on studies of humans and animals exposed to cumene, naphthalene or monomethyl-naphthalenes, but data concerning the rate and extent of absorption are limited. Animal studies indicate that these indicator compounds and their metabolites are widely distributed following absorption and that urinary excretion of metabolites is the primary route of elimination. Metabolism of cumene, naphthalene, and methyl naphthalenes involves *aromatic ring oxidation* (especially for naphthalene)-forming epoxide, alcohol, dihydrodiol, and quinone derivatives that can be conjugated to glutathione, glucuronic acid, or sulfate-and *oxidation of the alkyl side groups* (i.e., in cumene or methyl naphthalenes)-forming alcohol and carboxylic acid derivatives that can be conjugated to glucuronic acid or amino acids (ATSDR 1995e; EPA 1987a, 1997b).

Hydrocarbons in the aromatic $EC_{>16}$ –EC_{35} fraction may be absorbed to varying extents following inhalation, oral, or dermal exposure, depending on the lipophilicity and molecular size of the compound and the vehicle of administration, as indicated by studies of humans exposed to workplace-air complex

mixtures containing PAHs (i.e., hydrocarbons with more than two 5- or 6-carbon aromatic rings) and studies of animals exposed to individual PAHs by inhalation, oral administration, or dermal application. Increasing lipophilicity of vehicles or of the PAH compound tends to increase absorption, whereas adsorption to particles of increasing size (especially for inhalation exposure) or increasing molecular weight of the PAH compound tends to decrease absorption. Following absorption, PAHs are widely distributed to tissues and organs and eliminated by urinary and biliary excretion of metabolites. Metabolism of PAHs involves the production of arene oxides, phenols, quinones, dihydrodiols (i.e., diols), phenol-diols, and diol-epoxides, and the conjugation of these oxidized intermediates to glutathione, glucuronic acid or sulfate. Reactive metabolic intermediates, including stereospecific isomers of arene oxides and diol-epoxides, are thought to cause the genotoxic and carcinogenic effects produced by carcinogenic PAHs (ATSDR 1995f).

Hydrocarbons in the aliphatic EC_5 –EC_8 fraction may be readily absorbed in the lungs, as indicated by studies of humans and animals exposed to *n*-hexane, but absorption by the oral and dermal route is not well characterized. Aspiration to the lungs can occur following ingestion of hydrocarbons in this fraction. Absorbed *n*-hexane, based on determined partition coefficients in human and animal tissues, is expected to be widely distributed to tissues and organs with preferential partitioning into fatty tissues and well perfused tissues. Studies with humans and animals indicate that *n*-hexane is oxidatively metabolized to alcohol, ketone, carboxylic acid, dihydrodiol, and diketone derivatives, predominately in the liver. Urinary excretion of metabolites and, to a lesser extent, exhalation of unchanged *n*-hexane are the predominant means of elimination with low-level exposure, whereas exhalation of unchanged compound becomes a more important elimination pathway with high exposures (ATSDR 1999b).

Hydrocarbons in the aliphatic $EC_{>8}$ –EC_{16} fraction may be readily absorbed in the lungs, widely distributed to tissues with preferential distribution and accumulation occurring in fatty tissues, and slowly eliminated from fatty tissue, as indicated by studies of humans exposed by inhalation to a mixture of C_{10}-C_{12} alkanes (“white spirit”) and studies of rats exposed by inhalation to single alkanes or cycloalkanes in the C_6-C_{10} range. Results from these studies suggest that metabolism of hydrocarbons in this fraction, especially following distribution to fatty tissue, may be slow relative to aromatic hydrocarbons. Aspiration to the lungs may occur following ingestion of hydrocarbons in this fraction, especially those at the lower end of the ranges of molecular weight and viscosity for the fraction. Studies with rats indicate

that percentage absorption of ingested aliphatic hydrocarbons decreases with increasing carbon number from about 60% for C_{l4} compounds to 5% or less for hydrocarbons with 228 carbons.

Hydrocarbons in the aliphatic $EC_{>16}$ –EC_{35} fraction may be poorly absorbed, regardless of the route of exposure, preferentially distributed to the liver and fatty tissues, slowly metabolized to fatty acids or triglycerides, and slowly excreted in the feces via the bile and as urinary metabolites, as indicated by studies with animals exposed to food-grade mineral oil or motor oil (ATSDR 1997b). The common presence of lipogranulomata in human autopsies (benign structures in human liver and spleen tissue which are composed of lipoid droplets surrounded by lymphocytes and macrophages and caused by dietary exposure to mineral oils) is consistent with the concept that aliphatic hydrocarbons in this fraction are slowly metabolized.

6.4.1 Absorption

6.4.1.I Inhalation Exposure

Aromatic EC_5 –EC_9 Fraction. Studies with humans and animals are available for each of the BTEXs; these studies indicate that BTEX compounds are rapidly and efficiently absorbed following inhalation exposure. Published retention percentages for inspired BTEXs in human studies range from approximately 30% to 70-80% (see ATSDR 1994, 1995d, 1997a, 1999a).

Aromatic $EC_{>9}$ –EC_{16} Fraction. Studies measuring the rate and extent of absorption in humans or animals following inhalation exposure to naphthalene or the monomethyl naphthalenes were not available, but observations of systemic health effects in humans and animals provide qualitative evidence of absorption of these indicator compounds (ATSDR 1995e). Studies of humans following inhalation exposure to isopropylbenzene (cumene) indicated a retention percentage of about 50% (EPA 1987a, 1997b).

Aromatic $EC_{>16}$ –EC_{35} Fraction. Studies directly measuring the rate and extent of absorption in humans or animals following inhalation exposure to PAHs were not available, but measurement of the appearance of radioactivity in blood, tissues, and excreta within hours of exposure of animals to airborne, radioactively labeled benzo(a)pyrene indicate that rapid absorption can occur. Particle size and vehicle are expected to influence the absorption of inhaled PAHs, as indicated by measurements of lung clearance

following inhalation exposure of rats to benzo(a)pyrene adsorbed onto particles of differing sizes and measurements of excretion rates in rats following intratracheal instillation of benzo(a)pyrene in various vehicles (ATSDR 1995f)

Aliphatic EC_5 –EC_8 Fraction. Studies with humans exposed to vapors of *n*-hexane indicate that 20-25% of inhaled compound is absorbed and retained (ATSDR 1999b). In studies with rats exposed by inhalation, 12 hours/day for 3 days, to 100 ppm single hydrocarbons in the C_6-C_{10} alkane series (*n*-hexane through *n*-decane) and a C_6-C_{10}, naphthene series (cyclohexane, methylcyclohexane, dimethylcyclohexane, trimethylcyclohexane and *t*-butylcyclohexane), absorption was demonstrated by the measurement of concentrations of hydrocarbons in blood, brain, liver, kidneys, and fat (Zahlsen et al. 1992). Within each series, tissue concentrations (μmol/kg) generally increased with increasing carbon number.

Aliphatic $EC_{>8}$ –EC_{16} Fraction. Hydrocarbons in this fraction may be readily absorbed following inhalation, as indicated by studies of humans exposed to airborne mixtures of mostly C_{10}-C_{12} hydrocarbons and by the studies of rats exposed to single hydrocarbons conducted by Zahlsen et al. (1992).

For human volunteers exposed by inhalation to 100 ppm white spirit for 3 hours, a mean pulmonary uptake of 392 mg white spirit was measured, based on concentrations of white spirit in inspiratory and expiratory air (Pedersen et al. 1987). Following exposure to the same concentration, 6 hours/day for 5 consecutive days, the mean pulmonary uptake was 3,464 mg white spirit. The test material was a mixture of aliphatic hydrocarbons containing 99% linear and branched alkanes (0.99% C_8-C_9, 15% C_{10}, 39% C_{11}, and 44% C_{12}), and 1% C_9-C_{10} cycloalkanes.

Absorption of inhaled hydrocarbons in the lower range of this fraction was demonstrated by detection of hydrocarbons in blood, brain, liver, kidneys, and fat in rats following exposure to single hydrocarbons (C_6 -C_{10}, *n*-alkanes [*n*-hexane through *n*-decane] and C_6-C_{10}, naphthenes [cyclohexane, methylcyclohexane, dimethylcyclohexane, trimethylcyclohexane, and *t*-butylcyclohexane]) at 100 ppm, 12 hours/day, for 3 days (Zahlsen et al. 1992).

Aliphatic $EC_{>16}$ –EC_{35} Fraction. Studies measuring the rate and extent of absorption of aliphatic hydrocarbons in this fraction were not located, but animal studies with mineral oil aerosols suggest that

absorption is not rapid and lung clearance may be mediated by macrophages. Mice, rats, and rabbits exposed to aerosols of diesel-engine lubricating oil for up to 343 days showed oil in alveolar macrophages, mediastinal lymph nodes, lymphatic channels of the lungs, and the pleura; in mice, concentrations (w/w) of oil were 0.13% in lungs and 0.03% in livers (ATSDR 1999b).

6.4.1.2 Oral Exposure

Aromatic EC_5 –EC_9 Fraction. Animal studies are available for each of the BTEXs, indicating that these compounds are rapidly and efficiently absorbed following oral exposure. Published absorption percentages for oral doses of BTEXs in animal studies range from about 80% to 97% (see ATSDR 1994, 1995d, 1997a, 1999a).

Aromatic $EC_{>9}$ –EC_{16} Fraction. No data regarding the extent or rate of absorption of ingested naphthalene or monomethyl naphthalenes were available, except for a report that 80% of an oral dose of 2-methyl naphthalene was recovered as metabolites in the urine of rats within 24 hours (ATSDR 1995e). Studies with animals indicate that orally administered isopropylbenzene (cumene) rapidly appeared in the blood and that 90% of the administered dose was accounted for in urinary metabolites (EPA 1987a, 1997b).

Aromatic $EC_{>16}$ –EC_{35} Fraction. Studies with animals following oral exposure to benzo(a)pyrene and other PAHs indicate that the extent of oral exposure to PAHs can vary depending on lipophilicity of the PAH compound and lipophilicity of the vehicle in which it is administered (ATSDR 1995f).

Aliphatic EC_5 –EC_8, $EC_{>8}$ –EC_{16}, and $EC_{>16}$ –EC_{35} Fractions. No studies were located regarding absorption of hydrocarbons in these fractions after oral exposure in humans. Studies in rats show that absorption of ingested aliphatic hydrocarbons (*n*-alkanes, isoparaffins, and naphthenes) is inversely related to molecular weight, ranging from complete absorption at the lower end of the molecular weight range to about 60% for C_{14} hydrocarbons, 5% for C_{28} hydrocarbons, and essentially no absorption for aliphatic hydrocarbons with >32 carbons (Albro and Fishbein 1970; Miller et al. 1996)

6.4.1.3 Dermal Exposure

Aromatic EC_5 –EC_9 Fraction. Studies with animals indicate that BTEXs are dermally absorbed, but to a lesser extent than absorption via inhalation or oral exposure, especially when exposure is to vapors of these compounds (as opposed to the liquids or liquid solutions) (see ATSDR 1994, 1995d, 1997a, 1999a).

Aromatic $EC_{>9}$ –EC_{16} Fraction. Data regarding the rate and extent of dermally administered isopropylbenzene (cumene), naphthalene, or monomethyl naphthalenes were restricted to observations of systemic effects in humans and animals following dermal exposure to these compounds (ATSDR 1995e; EPA 1987a, 1997b).

Aromatic $EC_{>16}$ –EC_{35} Fraction. Studies that monitored radioactivity in rat tissues, organs, and excreta following the dermal application of individual radiolabeled PAHs in an organic solvent measured absorption percentages in the approximate range of 50-80% (% of applied dose that was absorbed), but found that absorption percentages declined to less than 20% when soil particles were included in the applied material (ATSDR 1995f).

Aliphatic EC_5 –EC_8 Fraction. *In vitro* studies with human skin indicate that the permeability of *n*-hexane through skin was about l00-fold lower than the permeability of benzene, suggesting that hydrocarbons in this fraction may have a low potential for skin absorption (ATSDR 1999b).

Aliphatic $EC_{>8}$ –EC_{16} Fraction. No studies were located regarding absorption of hydrocarbons in this fraction after dermal exposure in humans or animals.

Aliphatic $EC_{>16}$ –EC_{35} Fraction. No studies were located that measured the rate or extent of dermal absorption of hydrocarbons in mineral oil or similar materials in animals or humans. Dermal absorption of hydrocarbons in this fraction, however, may be expected to be slow, based on studies with monkeys administered subcutaneous doses of radiolabeled mineral oil in an aqueous emulsion. Radioactivity remaining at the sites of injection accounted for 85-99% and 25-33% of the administered radioactivity, at 1 week and 10 months following injection, respectively (ATSDR 1997b).

6.4.2 Distribution

Aromatic EC_5 –EC_9 Fraction. Studies with humans and animals exposed predominately to vapors of individual BTEXs (there are fewer data for oral and dermal exposure) indicate that, following absorption, compounds in this fraction are widely distributed, especially to lipid-rich and highly perfused tissues (see ATSDR 1994, 1995d, 1997a, 1999a). Studies of rats exposed by inhalation to single hydrocarbons at 100 ppm, 12 hours/day, for 3 days found that C_6-C_{10} aromatics (benzene, toluene, xylene, trimethylbenzene, and *t*-butylbenzene), compared with C_6-C_{10}, *n*-alkanes (*n*-hexane through *n*-decane) and C_6-C_{10}, naphthenes (cyclohexane, methylcyclohexane, dimethylcyclohexane, trimethylcyclohexane, and *t*-butylcyclohexane), showed high concentrations (μmol/kg) in blood, low concentrations in organs, and a lower potential for accumulation in fat and other organs presumably due to faster metabolic disposition (Zahlsen et al. 1992).

Aromatic $EC_{>9}$ –EC_{16} Fraction. Studies of swine after oral exposure to naphthalene, rats after dermal exposure to naphthalene, and guinea pigs after oral exposure to 2-methyl naphthalene indicate that these compounds, and their metabolites, are distributed throughout tissues and organs following absorption (ATSDR 1995e). Studies with rats exposed to isopropylbenzene (cumene) by inhalation, oral administration, or intravenous injection indicated that absorbed isopropylbenzene (cumene) is distributed to many tissues and organs with some preferential distribution in fatty tissues (EPA 1987a, 1997b).

Aromatic $EC_{>16}$ –EC_{35} Fraction. Studies with animals exposed to individual radiolabeled PAHs by inhalation, oral administration, or dermal administration indicate that, following absorption, PAHs are widely distributed to tissues and organs (ATSDR 1995f). Studies with pregnant animals found that, following oral exposure to radiolabeled benzo(a)pyrene, placental levels of radioactivity were higher than levels in embryonic tissue, suggesting that benzo(a)pyrene does not readily cross the placental barrier (ATSDR 199%).

Aliphatic EC_5 –EC_8 Fraction. Determination of partition coefficients (blood:air and tissue:air) for *n*-hexane in human and rat tissues indicates that hydrocarbons in this fraction, once absorbed, will be widely distributed to tissues and organs with preferential distribution to fatty tissues and well perfused tissues (ATSDR 1999b). Asphyxia and chemical pneumonitis can be a health concern from ingestion of hydrocarbons in this fraction, due to aspiration to the lungs. The aspiration potential of ingested

hydrocarbons increases with decreasing viscosity; within the alkane series, C_6-C_{10}, viscosity decreases with decreasing molecular weight (Cavender 1994).

Studies of rats exposed by inhalation to single hydrocarbons at 100 ppm, 12 hours/day, for 3 days found that C_6-C_{10} *n*-alkanes (*n*-hexane through *n*-decane) and C_6-C_{10} naphthenes (cyclohexane, methylcyclohexane, dimethylcyclohexane, trimethylcyclohexane, and *t*-butylcyclohexane), compared with C_6-C_{10} aromatics (benzene, toluene, xylene, trimethylbenzene, and *t*-butylbenzene), generally showed low concentrations (μmol/kg) in blood, high concentrations in brain and other organs, and a high potential for accumulation in fat (Zahlsen et al. 1992). Within any of these three categories of hydrocarbons, hydrocarbon concentrations in tissues (blood, brain, kidney, liver and fat) generally increased with increasing carbon number (Zahlsen et al. 1992). Twelve hours after cessation of exposure, concentrations of alkanes and naphthenes in fat and brain were 2- to 3-fold higher than concentrations of aromatics, suggesting faster metabolic disposition for the aromatics.

Aliphatic $EC_{>8}$ –EC_{16} Fraction. Studies of rats exposed by inhalation to individual C_6-C_{10}, *n*-alkanes and cycloalkanes indicate that hydrocarbons in this fraction are distributed widely to tissues and organs after absorption and can accumulate in fat (Zahlsen et al. 1992). Aspiration to the lungs can occur following ingestion of hydrocarbons in this fraction (Cavender 1994). Following absorption from the gastrointestinal tract, smaller molecular weight aliphatic hydrocarbons and/or their metabolites are transported in the body via the blood and the lymph system, whereas larger molecular weight aliphatic hydrocarbons may be distributed predominately via the lymph system (see for review Albro and Fishbein 1970; Miller et al. 1996).

Aliphatic $EC_{>16}$ –EC_{35} Fraction. Lung accumulation of hydrocarbons from this fraction is of concern with prolonged or high-level exposure to aerosols or ingestion, as indicated by numerous case reports of lipoid pneumonia in humans exposed to mineral oil through intranasal application of liquid petrolatum in medicinal nose drops and by a case of lipoid pneumonia in a child who ingested a 5-10 mL dose of mineral oil automobile transmission fluid (ATSDR 1997b). Following absorption, hydrocarbons in this fraction may be expected to accumulate to some degree in liver and fatty tissues, as indicated by the observation that, 24 hours after administration of an oral dose of tritiated mineral oil to rats, concentrations of tritiated mineral oil were about 7-fold greater in fatty tissues and liver than in kidney and brain (ATSDR 1997b). Lipogranulomata (clusters of lipoid droplets surrounded by lymphocytes and

macrophages) are commonly found in human autopsies, particularly in liver, spleen, and abdominal lymph nodes (Miller et al. 1996; Wanless and Geddie 1985). These structures are associated with dietary exposure to mineral oils and waxes, and are considered a benign response without adverse consequences (Miller et al. 1996; Wanless and Geddie 1985).

6.4.3 Metabolism

Aromatic EC_5 –EC_9 Fraction. As indicated by studies with humans and animals exposed to individual BTEXs, compounds in this fraction may be expected to be metabolized via cytochrome P-450 oxidases, either at carbons in the aromatic ring or in alkyl side groups, to metabolic intermediates that can be conjugated with glucuronides, sulfates, glutathione, or ammo acids (e.g., cysteine or glycine). The resultant oxidated metabolites or conjugated metabolites are more water-soluble than parent compounds and are subject to urinary or, in some cases, biliary excretion. Metabolism of the BTEXs can represent both a detoxification process (e.g., enhancement of the formation and excretion of hippuric acid can counteract the acute neurotoxicity of toluene in animals) and a toxification process (e.g., cancer and hematopoietic effects from chronic exposure to benzene appear to be caused by reactive metabolic intermediates) (see ATSDR 1994, 1995d, 1997a, 1999a).

Aromatic $EC_{>9}$ –EC_{16} Fraction. Studies with animals following oral, intraperitoneal, or subcutaneous administration of naphthalene or 2-methyl naphthalene indicate that ring oxidation occurs via an initial epoxide intermediate that subsequently is converted to alcohol, dihydrodiol and quinone derivatives, some of which are conjugated to glutathione, glucuronic acid, or glycine, and that the presence of alkyl side groups presents another site for oxidation and conjugation (ATSDR 1995e). Naphthol and naphthoquinone derivatives have been detected in the urine of humans following exposure to naphthalene (ATSDR 1995e). Studies with animals exposed to isopropylbenzene (cumene), and with *in vitro* animal preparations, indicate that cumene is predominately oxidized at the l- or 2-carbon of the propyl side group to form alcohol or carboxylic acid derivatives that are conjugated predominately to glucuronic acid (EPA 1987a, 1997b). A study that analyzed urinary metabolites in humans following acute inhalation exposure to cumene provided supporting data (EPA 1987a).

Aromatic $EC_{>16}$ –EC_{35} Fraction. *In vitro* studies with human tissues and *in vitro* and *in vivo* animal studies with benzo(a)pyrene and other PAHs indicate that compounds in this TPH fraction will undergo oxidative metabolism involving the production of arene oxides, phenols, quinones, dihydrodiols (i.e., diols),

phenol-diols, and diol-epoxides (catalyzed by enzyme systems including cytochrome P-450 oxidases and epoxide hydrolase), and the conjugation of these intermediates to glutathione, glucuronic acid, or sulfate (ATSDR 1995f). Metabolism of PAHs facilitates both the elimination of more water soluble metabolites and the production of reactive intermediates (e.g., stereospecific isomers of arene oxides and diol-epoxides) thought to be responsible for the mutagenic and carcinogenic activity of carcinogenic PAHs (ATSDR 1995f).

Aliphatic EC_5 –EC_8 Fraction. Examination of urinary metabolites in humans and rats after exposure to *n*-hexane indicates that hydrocarbons in this fraction may be oxidatively metabolized via cytochrome P-450 oxidases to several alcohol, ketone, and carboxylic acid derivatives. Based on studies of urinary metabolites after exposure to *n*-hexane, proposed metabolites include l-, 2-, and 3-hexanol, 2-hexanone, 5-hydroxy-2-hexanone, 2,5-hexanedione, and hexanoic acid (ATSDR 1999b).

Aliphatic $EC_{>8}$ –EC_{16} Fraction. Hydrocarbons in this fraction are oxidatively metabolized to fatty acids and alcohols, apparently mediated by cytochrome P-450 isozymes (see Miller et al. 1996 for review). Studies regarding the metabolism of hydrocarbons in this fraction in humans or animals provide suggestive evidence that metabolism may be slow. In a study of humans exposed to 100 ppm white spirit 6 hours/day for 5 days (white spirit is a mixture comprised predominately of C_{10}-C_{12}, linear and branched alkanes), only minor differences were observed in the GC-MS spectrum of hydrocarbons in biopsied fatty tissue, than in the spectrum of hydrocarbons in the test material (Pedersen et al. 1984). In rats exposed by inhalation to single C_6-C_{10}, alkanes, cycloalkanes, or aromatic hydrocarbons at 100 ppm, 12 hours/day for 3 days, concentrations of alkanes and cycloalkanes were 2- to 3-fold higher than concentrations of aromatics 12 hours after cessation of exposure, suggesting that aliphatic hydrocarbons in this fraction may be metabolized more slowly than aromatic hydrocarbons of equivalent molecular weight (Zahlsen et al. 1992).

Aliphatic $EC_{>16}$ –EC_{35} Fraction. Aliphatic hydrocarbons in this fraction are not expected to undergo extensive metabolism in animals or humans. In monkeys, 2 days after intramuscular injection of a mineral oil emulsion with a radiolabeled C_{16} hydrocarbon (*n*-hexanedecane), substantial portions (30-90s) of radioactivity in various tissues existed as unmetabolized *n*-hexanedecane. The remainder of the radioactivity was found as phospholipids, free fatty acids, triglycerides, and sterol esters. No radioactivity was found in water-soluble fractions (ATSDR 1997b). The common presence of lipogranulomata in

human autopsies and the widespread dietary exposure to mineral oils and waxes (Wanless and Geddie 1985) are consistent with the concept that aliphatic hydrocarbons in this fraction are slowly metabolized.

6.4.4 Elimination and Excretion

Aromatic EC_5 –EC_9 Fraction. Studies with humans and animals exposed by various routes to BTEXs, indicate that compounds in this fraction may be expected to be eliminated predominately by urinary excretion of metabolites and to lesser degrees by exhalation of unchanged parent compound or biliary excretion of metabolites (see ATSDR 1994, 1995d, 1997a, 1999a).

Aromatic $EC_{>9}$–EC_{16} Fraction. Data from studies with animals exposed by several routes to naphthalene, monomethyl naphthalenes and isopropylbenzene (cumene) indicate that urinary excretion of metabolites represents the predominant pathway of elimination for these compounds. Detection of urinary metabolites in humans exposed to naphthalene or cumene provide supporting evidence (ATSDR 1995e; EPA 1987a, 1997b).

Aromatic $EC_{>16}$ –EC_8 Fraction. Studies with animals exposed by inhalation, and by oral, dermal, or parenteral administration, indicate that PAHs are eliminated by urinary and biliary excretion of metabolites (ATSDR 1995f).

Aliphatic EC_5 –EC_8 Fraction. Studies with humans and animals exposed to *n*-hexane suggest that hydrocarbons in this fraction, under low-exposure conditions, may be eliminated predominately as urinary metabolites and to a lesser extent in exhaled air as unchanged compound. Studies with rats indicate that the importance of exhalation of unchanged hexane as an elimination pathway increased from about 12% to 62% of body burden after inhalation exposure to 500 ppm and 10,000 ppm, respectively (ATSDR 1999b).

Aliphatic $EC_{>8}$ –EC_{16} Fraction. Results from studies with humans exposed by inhalation to white spirit (a mixture of C_{10}-C_{12} aliphatic hydrocarbons) suggest that hydrocarbons in this fraction are slowly eliminated following distribution to fatty tissues (Pedersen et al. 1984). Immediately after 5 consecutive days of 6-hour daily exposure to 100 ppm white spirit, the mean concentration of white spirit in fatty tissue was 41.1 mg/kg fat; approximately 60 exposure-free hours later, mean fatty tissue concentrations had declined by only 23% to 3 1.7 mg/kg fat. No studies were located regarding the routes of excretion for hydrocarbons in this fraction in humans or animals.

Aliphatic $EC_{>16}$ –EC_{35} Fraction. Hydrocarbons in this fraction may be expected to be eliminated predominately in the feces, based on experiments with rats given oral or intraperitoneal doses of tritiated mineral oil. With oral exposure, 90% of administered radioactivity appeared rapidly (within 2 days) in the feces, predominately as unchanged mineral oil; less than 10% of administered radioactivity appeared in the urine within 2 days of administration. With intraperitoneal exposure, radioactivity appeared more slowly in the feces (11% of administered radioactivity appeared in the feces within 8 days of dosing); urinary excretion of metabolites, within 8 days of dosing, represented about 8% of administered radioactivity (ATSDR 1997b).

6.4.5 Physiologically Based Pharmacokinetic (PBPK)/Pharmacodynamic (PD) Models

No studies were located regarding the development of PBPK/PD models for complex mixtures of TPH in general.

Verhaar et al. (1997), however, recently reported on progress in developing PBPK/PD models for use in assessing human health risks from exposure to JP-5, a Navy Jet petroleum fuel containing a complex mixture of hydrocarbons in the C_9C_{18}, range. Verhaar et al. (1997) noted that their in-progress development of a PBPK/PD model for JP-5 is focused on the prediction of kinetics of JP-5 components in relevant tissues after acute inhalation exposure and the resultant toxicity (neurological effects linked to the dissolution of xenobiotic chemicals in the membrane of nerve cells). Verhaar et al. (1997) discussed how the development of PBPK/PD model(s) for complex mixtures involves:

(1) determining a lumping scheme to be used (in which similar mixture components are grouped [i.e., lumped] into a *pseudocomponent* for which necessary chemical parameters such as tissue partition coefficients are estimated), based on knowledge of the mixture's chemical composition, the route and duration of exposure that is of interest, and the mixture's toxicological effect(s) and mechanism of action (a lumping scheme based on the octanol-water partition coefficients of components was chosen for JP-5);

(2) formulating PBPK/PD model(s) with physiological compartments, reaction kinetic equations, and mass transfer equations that are appropriate to the toxicological effect(s) of concern (a brain compartment, including a pharmacodynamic subroutine, was proposed to be included in the PBPK/PD model for JP-5);

(3) determining whether there is enough information to include interactive effects between *pseudocomponents* in the model(s); and

(4) using quantitative structure-activity relationships (QSAR) to estimate necessary model parameters for *pseudocomponents* such as tissue-blood and air-blood partition coefficients, and metabolic rate constants.

The approach discussed by Verhaar et al. (1997) suggests that development of PBPK/PD models to use in assessing health risks from TPH will require similar focusing on relevant lumping schemes, exposure pathways and durations, and toxicological effects and mechanisms of action. Thus, it is likely that a PBPK/PD model developed to aid in the assessment of potential cancer risk from chronic exposure to TPH may substantially differ from a PBPK/PD model for assessing risk for potential neurological effects from acute exposure to TPH.

6.5 MECHANISM OF ACTION

Because TPH is a broadly defined entity consisting of complex mixtures of hydrocarbons of varying chemical composition (due to differences in original petroleum products and differential, time-dependent, fate and transport of components within any particular TPH mixture), this section discusses available information for components and petroleum products corresponding to the transport fractions of TPH. Limited additional information regarding the more heterogenous whole petroleum products can be found in the ATSDR toxicological profiles and other assessments of these products referenced in Section 6.3. In general, however, there is little information regarding mechanisms for these heterogenous products. The discussions of mechanisms in these documents often deal with the individual constituents, including additives and impurities that are not petroleum hydrocarbons, and with hydrocarbon mixtures that are similar to portions of the product.

6.5.1 Pharmacokinetics Mechanisms

Absorption. Available data suggest that hydrocarbons in the aliphatic EC_5-EC_5 and aromatic EC_5-EC_9 fractions may be more readily absorbed by the lungs, gastrointestinal tract, and skin than hydrocarbons in the aliphatic or aromatic hydrocarbons in larger molecular weight fractions. This

difference is due to their smaller molecular size and the presumed dependence of absorption of hydrocarbons on diffusion or facilitated diffusion.

Distribution, Storage and Excretion. Hydrocarbons in each of the aliphatic and aromatic fractions are expected to be distributed throughout tissues and organs following absorption. Preferential distribution to fatty tissues occurs especially with aliphatic hydrocarbons. Ingested or inhaled volatile aliphatic and aromatic hydrocarbons in the EC_5-EC_8 and EC_5-EC_9 fractions can be eliminated in exhaled breath as unchanged parent compound. Metabolic elimination of aromatic hydrocarbons in each EC fraction predominately occurs via oxidative metabolic pathways involving initial oxidation by cytochrome P-450 isozymes and conjugation to more water-soluble compounds such as glutathione and glucuronic acid. Some studies in animals suggests that aliphatic hydrocarbons (especially in the $EC_{>8}$-EC_{16} and $EC_{>16}$-EC_{35} fractions) may be metabolized more slowly than aromatic hydrocarbons. Metabolites of both aliphatic and aromatic hydrocarbons are excreted in urine and in feces via biliary excretion.

Route-dependent Toxicity. Ingested aliphatic hydrocarbons in the EC_5-EC_8 and $EC_{>8}$-EC_{16} fractions are aspirated to the lungs and can lead to pulmonary irritation, edema, and pneumonia. Materials with low viscosity (in the range of 30-35 centipoise) present an extreme aspiration risk, whereas those with high viscosity (150-250 centipoise) present very low aspiration risk (Snodgrass 1997).

6.5.2 Mechanisms of Toxicity

Central nervous system (CNS) depression caused by acute inhalation exposure to volatile aliphatic and aromatic petroleum hydrocarbons is generally thought to occur when the lipophilic parent hydrocarbon dissolves in nerve cell membranes and disrupts the function of membrane proteins by disrupting their lipid environment or by directly altering protein conformation. Oxidative metabolism of CNS-depressing hydrocarbons reduces their lipophilicity and represents a process that counteracts CNS-depression toxicity. More detailed information on this mechanism of toxicity can be found in ATSDR profiles on toluene (ATSDR 1994), ethylbenzene (ATSDR 1999a), and xylene (ATSDR 1995d).

Pulmonary irritation and pneumonia from inhalation and oral exposure to complex mixtures of petroleum hydrocarbons such as gasoline and kerosene are thought to involve direct parent hydrocarbon interaction with nerve cell membranes resulting in bronchoconstriction and dissolution into membranes of lung

parenchyma resulting in a hemorrhagic exudation of proteins, cells, and fibrin into alveoli (ATSDR 1998b; Klaassen 1996).

In contrast, metabolic bioactivation, mediated by pathways involving cytochrome P-450 isozymes, is thought to be responsible for hemolytic anemia and leukemia from exposure to benzene (ATSDR 1997a) genotoxic effects and cancer from exposure to carcinogenic PAHs (ATSDR 19950; hemolytic anemia, ocular effects, and lung effects from naphthalene and methyl naphthalenes (ATSDR 1995e); peripheral neuropathy from *n*-hexane (ATSDR 1999b); lung effects from ethylbenzene (ATSDR 1999a); and $\alpha_{2\mu}$-globulin nephropathy (which is unique to male rats) from hydrocarbons in gasoline (ATSDR 1995a).

6.5.3 Animal-to-Human Extrapolations

Rats and mice are much less sensitive than humans to the hemolytic effects of naphthalene. The dog appears to be a better model for humans for this effect (ATSDR 1995e).

Inhalation or oral exposure to a number of the individual constituents of the TPH fractions (particularly branched-chain alkanes) and also the petroleum products whose composition is similar to these fractions (e.g., JP-5, JP-7, and the dearomatized streams) induces a hydrocarbon-related nephropathy unique to male rats (ATSDR 1995c, 1995g, 1998b; TPHCWG 1997c). This lesion involves the formation of hyaline droplets in the cytoplasm of the proximal tubule cells of the cortex. The hyaline droplets contain high concentrations of the protein $\alpha_{2\mu}$-globulin, a protein found in male rats but not in humans. A likely mechanism for this accumulation is the slowing of the degradation of $\alpha_{2\mu}$-globulin as a result of binding with specific substances, such as petroleum hydrocarbons or their metabolites. Single cell necrosis and exfoliation of the proximal tubular epithelium occurs, and the tubules near the cortico-medullary junction become dilated and are eventually filled with coarsely granular casts and necrotic debris. Regenerative tubule cell proliferation and mineralization of the renal papillar tubules occurs with continued exposure. The nephropathy induced by accumulation of this protein has not been noted in female rats, in male rats that lack the ability to synthesize $\alpha_{2\mu}$-globulin, or in other species. Thus, it does not appear that the nephrotoxicity attributable to the $\alpha_{2\mu}$-globulin syndrome observed in male rats is relevant to humans.

Food grade and medicinal mineral oils which correspond to the aliphatic $EC_{>16}$-EC_{35} fraction of TPH produce liver granulomas in F344 rats. These granulomas are reactive, with associated inflammation and occasional parenchymal cell necrosis. The inflammatory effects are not seen in dogs, mice, or Long-Evans

or Sprague-Dawley rats fed comparable doses of similar mineral oils, according to TPHCWG (1997c). In addition, humans, who are exposed to mineral oils in the diet and by intentional ingestion of medicinal mineral oils, develop granulomas, but without evidence of inflammation or significant liver dysfunction. Whether the exposure levels for humans are comparable to those tested in experimental animals is not known. Nevertheless, the issue has been raised that F344 rats may be uniquely predisposed to the development of inflammatory granulomatous lesions, and that this difference in sensitivity may justify use of a smaller uncertainty factor in extrapolating from the F344 rat to humans (TPHCWG 1997c).

6.6 SELECTION OF FRACTION-SPECIFIC HEALTH EFFECTS CRITERIA

6.6.1 Overview

The focus of this section is the selection, when possible, of appropriate MRLs for the assessment of health effects of the aromatic and aliphatic fractions of TPH. Approaches to cancer assessment are also discussed. The TPH fractions are environmental transport fractions, as suggested by the TPHCWG (1997c), with a slight modification to include all the BTEXs in a redefined aromatic EC_5-EC_9 fraction.

Other agencies have addressed the problem of selection of health effects criteria for fractions or representative constituents of TPH (ASTM 1995; Hutcheson et al. 1996; TPHCWG 1997c), and their approaches were carefully evaluated during the preparation of this profile, as discussed in Sections 6.1 and 6.2. Nevertheless, ATSDR's concerns and mandate encompass a broader range of exposure periods than those of the other agencies, and ATSDR health criteria are developed somewhat differently and for a slightly different purpose. These issues were discussed in Section 6.1 and 6.2.

Tables 6-l and 6-2 summarize the suggested fraction-specific MRLs for inhalation and oral exposure. These fraction-specific MRLs are provisional values, reflecting the uncertainty inherent in this approach (see Section 6.6.2 for a more complete discussion). As with any ATSDR MRL, the MRLs in Tables 6-l and 6-2 are intended to serve as health guidance values and are not to be used to define clean-up or action levels. Information listed in brackets in Table 6-2 is from sources other than ATSDR toxicological profiles. This information indicates potentially sensitive end points but does not have the same level of confidence as information from the ATSDR toxicological profiles. Additional details and tables listing all the candidate MRLs and relevant cancer assessments are presented in Section 6.6.2. Chapter 7 also

Table 6-1. Fraction-Specific Provisional Inhalation MRLs and Critical Effects

Fraction	Indicator or surrogate compound or mixture	Acute MRL		Intermediate MRL		Chronic MRL	
		ppm	Effect	ppm	Effect	ppm	Effect
Aromatic							
EC_5–EC_9: Indicator Compounds	Benzene	0.05	Immunological/ lymphoreticular	0.004	Neurological	—	—
	Toluene	3	Neurological	—	—	1	Neurological
	Ethylbenzene	—	—	0.2	Developmental	—	—
	Xylene	1	Neurological	0.7	Developmental (neurological)	0.1	Neurological
$EC_{>9}$–EC_{16}	Naphthalene	—	—	—	—	0.002	Respiratory
$EC_{>16}$–EC_{35}	No data	—	—	—	—	—	—
Aliphatic							
EC_5–EC_8	n-Hexane	—	—	—	—	0.6	Neurological
$EC_{>8}$–EC_{16}	JP-5 and 8 JP-7	—	—	3 mg/m^3	Hepatic	0.3 mg/m^3	Hepatic
$EC_{>16}$–EC_{35}	No data	—	—	—	—	—	—

EC = Equivalent Carbon Number Index; MRL = minimal risk level

Source: Appendix A. MRLs and critical effects are summarized in Appendix A of this profile. Additional information is available in the profile for each compound (e.g., ATSDR. 1999b. Toxicological profile for hexane).

Table 6-2. Fraction-Specific Provisional Oral MRLs and Critical Effects[a]

Fraction	Indicator or surrogate compound or mixture	Acute MRL		Intermediate MRL		Chronic MRL	
		mg/kg/day	Effect	mg/kg/day	Effect	mg/kg/day	Effect
Aromatic							
EC_5–EC_9: Indicator Compounds	Benzene	—	—	—	—	—	—
	Toluene	0.8	Neurological	0.02	Neurological	—	—
	Ethylbenzene	—	—	—	—	—	—
	Xylene, mixed	—	—	0.2	Renal	—	—
	Xylene, *m*-	—	—	0.6	Hepatic	—	—
	Xylene, *p*-	1	Neurological	—	—	—	—
$EC_{>9}$–EC_{16}	Naphthalene	0.05	Neurological	0.02	Hepatic	—[a]	—
$EC_{>16}$–EC_{35}	Fluorene, fluoranthene	—	—	0.4	Hepatic	—	—
Aliphatic							
EC_5–EC_8	No data	—	—	—	—	—	—
$EC_{>8}$–EC_{16}	No ATSDR MRLs [Dearomatized petroleum streams][b]	—	—	——	[Hepatic][b]	—	—
$EC_{>16}$–EC_{35}	No ATSDR MRLs [Mineral oils C_{15}–C_{37}][b]	—	—	—	[Hepatic][b]	—	—

[a] No chronic MRL appears suitable for the assessment of health effects of the aromatic $EC_{>9}$-EC_{16} fraction as a whole, but a chronic MRL of 0.07 mg/kg/day is available for 1-methylnaphthalene.

[b] Critical effects are listed in brackets for mixtures that are not the subjects of ATSDR toxicological profiles, but have been evaluated by other agencies for the purpose of deriving health effects criteria. These health effects are shown only to indicate potentially sensitive endpoints of exposure to that fraction, and do not have the same level of confidence as an ATSDR assessment.

EC = Equivalent Carbon Number Index; MRL = minimal risk level

Source: Appendix A. MRLs and critical effects are summarized in Appendix A of this profile. Additional information is available in the profile for each compound (e.g., ATSDR. 1999b. Toxicological profile for hexane).

presents MRLs for constituents and whole petroleum products and health effects criteria developed by other agencies (EPA and TPHCWG RfDs and RfCs).

6.6.2 Minimal Risk Levels, Critical Effects, and Cancer Assessments for Fractions of TPH

The information in the following text is taken from the references cited in the tables that accompany the text. For the sake of readability, the references will not be cited in the text. Additional health effects information is available in the pertinent toxicological profiles (ATSDR 1994, 1995c, 1995d, 1995e, 1995f, 1995g, 1997a, 1998b, 1999a, 1999b), TPHCWG (1997c), EPA references cited in the tables including EPA (1998b), and in Section 6.2. In order to fill data gaps, some compounds, representative mixtures, or studies that have not been assessed in ATSDR toxicological profiles are listed, with the critical or sensitive effects as evaluated by other agencies (EPA and TPHCWG) shown in brackets. This was done to give a more complete picture of the potential health effects of fraction constituents, to aid in judging whether the available MRLs may be useful in assessing health effects of the entire fraction.

Aromatic EC_{5-9} Fraction: Indicator Compounds. This fraction consists of benzene, toluene, ethylbenzene and the xylenes (the BTEXs).

Inhalation Exposure. The available inhalation MRLs for each of the BTEXs, and the EPA cancer risk for benzene, can be used to assess the potential for health effects for each of these indicator compounds individually. This is consistent with current practice. These MRLs and their associated effects, as well as the EPA cancer assessments, are summarized in Table 6-3. Health effects that are common to the BTEXs are neurological effects. Developmental effects appear to be a sensitive effect of inhalation exposure to ethylbenzene and xylene. Benzene has hematological and immunological/lymphoreticular effects and is classified in EPA Group A (human carcinogen).

Oral Exposure. The oral MRLs for each of the BTEXs, and the EPA cancer risk for benzene, can be used to assess the potential for health effects for each of these compounds individually. No oral MRLs exist for ethylbenzene, but the limited oral data for this compound are reasonably similar to those for toluene. These MRLs and their associated effects, and the available EPA cancer assessments, are summarized in Table 6-4. Effects of oral exposure to these compounds are similar to those of inhalation exposure. In addition, renal and hepatic effects appear to be sensitive effects of xylene exposure.

Table 6-3. Inhalation MRLs, Critical Effects, and EPA Cancer Assessments for Aromatic EC_5–EC_9 Fraction

C	EC	Compound	ATSDR Toxicological Profile	Acute		Intermediate		Chronic		
				MRL		MRL		MRL		EPA Cancer WOE, risk per 1 ppm[a]
				ppm	Effect	ppm	Effect	ppm	Effect	
6	6.5	Benzene	1997a	0.05	Immunological/ lymphoreticular	0.004	Neurological	–	–	A, $2.7x10^{-2}$
7	7.58	Toluene	1994	3	Neurological	–	–	1	Neurological	D, NA
8	8.5	Ethylbenzene	1999a	–	–	0.2	Developmental	–	–	D, NA[b]
8	8.6–8.81	Xylene, mixed	1995d	1	Neurological	0.7	Developmental (neurological)	0.1	Neurological	D, NA

[a] EPA cancer WOE and risk are from the cited ATSDR toxicological profile and/or IRIS (EPA 1998b).

[b] EPA Classification in Group D (EPA 1998b) occurred prior to publication of chronic inhalation study of ethylbenzene (NTP 1996). ATSDR (1999a) notes that this classification is likely to change in the near future because the NTP study provides evidence of carcinogenicity (renal and testicular) in male rats and suggestive evidence in female rats and male and female mice.

C = carbon number; EC = Equivalent Carbon Number Index; MRL = minimal risk level; NA = not applicable; WOE = weight-of-evidence classification for carcinogenicity

Table 6-4. Oral MRLs, Critical Effects, and EPA Cancer Assessments for Aromatic EC_5 - EC_9 Fraction

C	EC	Compound	ATSDR Toxicological Profile	Acute		Intermediate		Chronic		
				MRL		MRL		MRL		EPA Cancer WOE, risk per mg/kg/day[a]
				mg/kg/day	Effect	mg/kg/day	Effect	mg/kg/day	Effect	
6	6.5	Benzene	1997a	–	–	–	–	–	–	A, $2.9x10^{-2}$[b]
7	7.58	Toluene	1994	0.8	Neurological	0.02	Neurological	–	–	D, NA
8	8.85	Ethylbenzene	1999a	–	–	–	–	–	–	D, NA[c]
8	8.6–8.81	Xylene, mixed	1995d	–	–	0.2	Renal	–	–	D, NA
8	8.6	Xylene, *-m*	1995d	–	–	0.6	Hepatic	–	–	–
8	8.61	Xylene, *p-*	1995d	1	Neurological	–	–	–	–	–

[a] EPA cancer WOE and risk are from the cited ATSDR toxicological profile and/or IRIS (EPA 1998b).

[b] Dose levels associated with excess cancer risks of 10^{-4}, 10^{-5}, 10^{-6}, and 10^{-7} have been calculated to be $3x10^{-3}$, $3x10^{-4}$, $3x10^{-5}$, and $3x10^{-6}$ mg/kg/day, respectively.

[c] EPA Classification in Group D (EPA 1998b) occurred prior to publication of a chronic inhalation study of ethylbenzene (NTP 1996). ATSDR (1999a) notes that this classification is likely to change in the near future because the NTP study provides evidence of carcinogenicity (renal and testicular) in male rats and suggestive evidence in female rats and male and female mice.

C = carbon number; EC = Equivalent Carbon Number Index; MRL = minimal risk level; NA = not applicable; WOE = weight-of-evidence classification for carcinogenicity

Aromatic $EC_{>9}$-EC_{16} Combined Fraction. The combined fraction consists of the following three fractions:

$EC_{>9}$-EC_{10} : a variety of alkylbenzenes (propyl-, methylethyl, trimethyl, and branched-chain butyl)

$EC_{>10}$-EC_{12}: a few alkylbenzenes (*n*-butyl-, *n*-pentyl-, a trimethyl-, and other multisubstituted), indans, and naphthalene

$EC_{>12}$-EC_{16}: a few longer chain and multi-substituted alkylbenzenes; biphenyls, methyl naphthalenes, and some smaller PAHs.

Inhalation Exposure. A chronic inhalation MRL is available for naphthalene; this MRL is listed in Table 6-5. There are no other inhalation MRLs for this fraction. All of the compounds in this fraction that have EPA carcinogenicity assessments have been classified in group D (not classifiable as to human carcinogenicity). Given the few health effects benchmarks available for the constituents of this fraction, and the general paucity of inhalation data for this fraction (see Section 6.2.2.1), selection of surrogate values for the combined fraction is problematic. Health effects that appear to be common to the compounds in this fraction are respiratory irritant effects, neurological effects, and renal effects, but it is not clear that they are common to all, or even that adequate investigation of respiratory or neurological effects was conducted for all compounds in the table. Based on some commonality of effect, the chronic MRL of 0.002 ppm for naphthalene could be adopted as a surrogate value for the combined fraction as a provisional measure. Great uncertainties are attendant on this selection, but the alternative is to disregard the potential for health effects of much of the mass of this fraction.

Oral Exposure. The only MRLs available for this fraction are acute and subchronic MRLs for naphthalene, an intermediate MRL for acenaphthene, and a chronic MRL for l-methyl naphthalene; these MRLs are listed in Table 6-6. Although more health effects data are available for oral exposure than for inhalation exposure to the constituents of this fraction, selection of surrogate values to use for oral exposure to this fraction is problematic. The acute and intermediate MRLs for naphthalene, 0.05 and 0.02 mg/kg/day, are equivalent to or lower than any other MRLs for this fraction, including the chronic MRL for l-methyl naphthalene. The compounds in this fraction tend to cause hepatic and renal effects. Naphthalene and l-methyl naphthalene have respiratory effects following oral exposure; it is expected that

Table 6-5. Inhalation MRLs, Critical Effects, and EPA Cancer Assessments for Aromatic $EC_{>9}$–EC_{16} Fraction

C	EC	Compound or mixture	ATSDR Toxicological Profile	Acute		Intermediate		Chronic		
				MRL		MRL		MRL		EPA Cancer WOE, risk per ppm[a]
				ppm	Effect	ppm	Effect[a]	ppm	Effect	
$EC_{>9}$-EC_{10}										
9	9.13	Isopropylbenzene (cumene)	–	–	–	–	[Renal and endocrine][b]	–	–	D, NA[b]
9 (8-10)	9.47–9.84 (8.81–0.52)	C_9 Aromatics: High flash aromatic naphtha[c]	–	–	–	–	–	–	[Hepatic and renal][c]	–
$EC_{>10}$-EC_{12}										
10	11.69	Naphthalene	1995e	–	–	–	–	0.002	Respiratory	D, NA[d]
$EC_{>12}$-EC_{16}										
12	15.06	Acenaphthylene	1995f	–	–	–	–	–	–	D, NA

[a] EPA cancer WOE and risk are from the cited ATSDR toxicological profile and/or IRIS (EPA 1998b). Critical effects are listed in brackets for mixtures or compounds that are not the subjects of ATSDR toxicological profiles, but have been evaluated by other agencies for the purpose of deriving health effects criteria. These health effects are shown only to indicate potentially sensitive endpoints of exposure to that fraction, and do not have the same level of confidence as an ATSDR assessment.

[b] EPA (1998b) concluded that the listed effect, which occurred in a 13-week inhalation study in rats (Cushman et al. 1995), was the critical effect.

[c] A mixture composed primarily of C_9 alkylbenzenes, with approximately 80% in the EC_9–EC_{10} range and the entire mixture (identified constituents) within the ranges shown in parentheses in the table. The major constituents of the mixture are trimethylbenzenes and methylethylbenzenes. According to the TPHCWG (1997b) the listed critical effect was seen in a 1-year inhalation study in rats (Clark et al. 1989).

[d] EPA Classification in Group D occurred prior to publication of a chronic inhalation study of naphthalene in mice (NTP 1992). A 1995 note added to the carcinogenicity file on IRIS indicates naphthalene may be more appropriately classified in Group C (EPA 1998b).

C = carbon number; EC = Equivalent Carbon Number Index; MRL = minimal risk level; NA = not applicable; WOE = weight-of-evidence classification for carcinogenicity

Table 6-6. Oral MRLs, Critical Effects, and EPA Cancer Assessments for Aromatic $EC_{>9}$–EC_{16} Fraction

C	EC	Compound	ATSDR Tox. Profile	Acute		Intermediate		Chronic		
				MRL		MRL		MRL		EPA Cancer WOE, risk per mg/kg/day[a]
				mg/kg/day	Effect	mg/kg/day	Effect[a]	mg/kg/day	Effect[a]	
$EC_{>9}$-EC_{10}										
9	9.13	Isopropylbenzene (cumene)	–	–	–	–	[Renal][b]	–	–	D, NA
9	9.62	1,3,5-Trimethyl-benzene	–	–	–	–	[Hepatic, renal, other][c]	–	–	–
$EC_{>10}$-EC_{12}										
10	11.69	Naphthalene	1995e	0.05	Neurological	0.02	Hepatic	–	–	D, NA[d]
10-11	11.69-12.99	Naphthalene/ methylnaphtha-lene mixture[e]	–	–	–	–	[Hepatic, endocrine, other][e]	–	–	–
$EC_{>12}$-EC_{16}										
11	12.99	1-Methyl-naphthalene	1995e	–	–	–	–	0.07	Respiratory	–
12	14.26	Biphenyl	–	–	–	–	–	–	[Renal][f]	D, NA
12	15.06	Acenaphthylene	1995f	–	–	–	–	–	–	D, NA
12	15.5	Acenaphthene	1995f	–	–	0.6	Hepatic	–	–	–

[a] EPA cancer WOE and risk are from the cited ATSDR toxicological profile and/or IRIS (EPA 1998b). Critical effects are listed in brackets for mixtures or compounds that are not the subjects of ATSDR toxicological profiles, but have been evaluated by other agencies for the purpose of deriving health effects criteria. These health effects are shown only to indicate potentially sensitive endpoints of exposure to that fraction, and do not have the same level of confidence as an ATSDR assessment.

[b] EPA (1997a, 1998b) concluded that the listed effect, which occurred in a 194-day oral study of isopropylbenzene in rats, was the critical effect.

[c] EPA (1996b) concluded that the listed effect, seen in a 90-day oral study in rats, was the critical effect.

[d] EPA Classification in Group D occurred prior to publication of chronic inhalation study of naphthalene in mice (NTP 1992). A 1995 note added to the carcinogenicity file on IRIS indicates naphthalene may be more appropriately classified in Group C (EPA 1998b).

[e] The TPHCWG (1997b) concluded that the listed effect, observed in an unpublished 13-week oral study of a mixture of naphthalene and methylnaphthalenes, was the critical effect. The composition of the mixture was not further specified. The above C and EC values assume the mixture contained naphthalene and *mono*methylnaphthalenes).

[f] According to EPA (1998b), the listed effect, seen in a lifetime oral study of 1,1,-biphenyl in rats, was the critical effect.

C = carbon number; EC = Equivalent Carbon Number Index; MRL = minimal risk level; NA = not applicable; WOE = weight-of-evidence classification for carcinogenicity

2-methyl naphthalene will as well. Neurological effects have been seen from acute exposure to naphthalene, and would also be expected with the alkyl benzenes, based on the inhalation data. Thus, there is some commonality in the health effects. Naphthalene appears to be one of the more toxic constituents of this fraction, so adoption of the MRLs for naphthalene as surrogate values for the entire mass of this fraction should be relatively protective. There is no chronic MRL for naphthalene, however, and the chronic MRL for l-methyl naphthalene (0.07 mg/kg/day) is similar to, but slightly higher than the intermediate MRL for naphthalene.

Aromatic $EC_{>16}$- EC_{35} Combined Fraction. The combined fraction consists of the following two fractions:

$EC_{>16}$- EC_{21}: anthracene, fluorene, phenanthrene, pyrene and other less well-known PAHs

$EC_{>21}$- EC_{35}: benz(a)anthracene; benzo(b)-, benzoCj)- and benzo(k)fluoranthene; benzo(g,h,i)perylene; benzo(a)- and benzo(e)pyrene; chrysene; dibenz(a,h)anthracene; fluoranthene; and indeno(1,2,3-c,d)pyrene, and other less well-known PAHs.

Inhalation Exposure. Very few health effects data for inhalation exposure and no inhalation MRLs are available for this fraction. Given the nonvolatile nature of these compounds, inhalation exposure as a result of contamination at hazardous waste sites would be anticipated to occur only through exposure to dust or particles containing PAHs.

Oral Exposure. The limited oral data for these PAHs indicate that hepatic effects are a common sensitive effect; renal effects have been seen with some. Intermediate MRLs of 0.4 mg/kg/day have been derived for fluorene and fluoranthene and of 10 mg/kg/day for anthracene; these are listed in Table 6-7. All of the commonly studied PAHs in the $EC_{>16}$-EC_{21} portion of the combined fraction have been classified in Group D (not classifiable as to human carcinogenicity). Many of the commonly studied PAHs in the $EC_{>21}$-EC_{35} portion of the combined fraction have been classified in Group B2 (probable human carcinogen). An intermediate MRL of 0.4 mg/kg/day was selected as a surrogate value for the combined fraction and should be applied to the non-carcinogenic PAHs in this fraction. A method for assessing the potential carcinogenic effects of these PAHs would be to use the EPA cancer risk levels for benzo(a)pyrene and the relative potency factors for the individual PAHs (Table 6-7).

Table 6-7. Oral MRLs, Critical Effects, and EPA Cancer Assessments for Aromatic $EC_{>16}$–EC_{35} Fraction[a]

C	EC	Compound	Acute		Intermediate		Chronic		
			MRL		MRL		MRL		EPA Cancer WOE, risk per mg/kg/day[b]
			mg/kg/day	Effect	mg/kg/day	Effect[b]	mg/kg/day	Effect	
$EC_{>16}$-EC_{21}									
13	17	Fluorene	–	–	0.4	Hepatic	–	–	D, NA
14	19	Phenanthrene	–	–	–	–	–	–	D, NA
14	19	Anthracene	–	–	10	Hepatic	–	–	D, NA
16	21	Pyrene	–	–		[Renal][c]	–	–	D, NA
$EC_{>21}$-EC_{35}									
16	22	Fluoranthene	–	–	0.4	Hepatic	–	–	D, NA
18	26	Benz[a]anthracene	–	–	–	–	–	–	B2, RP=0.145
18	27	Chrysene	–	–	–	–	–	–	B2, RP=0.0044
20	30	Benzo[b]fluoranthene	–	–	–	–	–	–	B2, RP=0.167
20	30	Benzo[k]fluoranthene	–	–	–	–	–	–	B2, RP=0.020
20	31	Benzo[a]pyrene	–	–	–	–	–	–	B2, 7.3[d]; RP=1
22	34	Dibenz[a,h]anthracene	–	–	–	–	–	–	B2, RP=1.11
22	34	Benzo[g,h,i]perylene	–	–	–	–	–	–	D, NA
22	35	Indeno[1,2,3-cd]pyrene	–	–	–	–	–	–	B2, RP=0.055

[a] All the compounds in this table are PAHs, and are included in the ATSDR toxicological profile on PAHs (ATSDR 1995f).

[b] EPA cancer WOE and risk are from the cited ATSDR toxicological profile and/or IRIS (EPA 1998b). Critical effects are listed in brackets for mixtures, compounds, or studies that are not the subjects of ATSDR toxicological profiles, but have been evaluated by other agencies for the purposes of deriving health effects criteria. These health effects are shown only to indicate potentially sensitive endpoints of exposure to that fraction, and do not have the same level of confidence as an ATSDR assessment.

[c] EPA (1997a, 1998b) concluded that the listed effect was the critical effect of pyrene, based on an unpublished oral 13-week study in mice (EPA 1995f). Although pyrene is included in the ATSDR toxicological profile on PAHs, this study was not cited (ATSDR 1995f). Therefore, it appears the study was not available to ATSDR for evaluation as a potential basis for an MRL.

[d] Dose levels associated with excess cancer risks of 10^{-4}, 10^{-5}, 10^{-6}, and 10^{-7} have been calculated to be $1x10^{-5}$, $1x10^{-6}$, $1x10^{-7}$, and $1x10^{-8}$ mg/kg/day, respectively.

C = carbon number; EC = Equivalent Carbon Number Index; MRL = minimal risk level; NA = not applicable; RP = Relative potency factor = the carcinogenic potency of this compound, relative to benzo[a]pyrene, as estimated by EPA (1993c) and reported by ATSDR (1995f). EPA (1993c) also reported relative potencies rounded to an order of magnitude and recommended that these rounded potencies be used because the quality of the data and the analysis do not support greater precision; WOE = weight-of-evidence classification for carcinogenicity

Aliphatic EC_5-EC_8 Combined Fraction. The combined fraction consists of the following two fractions:

EC_5- EC_6: *n*-pentane, *n*-hexane, dimethylbutanes, and methylpentanes, cyclopentane, some alkenes

$EC_{>6}$- EC_8: *n*-heptane, *n*-octane, some branched chain C_6-C_9 alkanes including trimethylpentanes, cyclohexane, methylcyclohexane, other cycloalkanes, some alkenes.

Inhalation Exposure. Only one inhalation MRL, a chronic MRL for *n*-hexane, is available for this combined fraction; this is listed in Table 6-8. *n*-Hexane produces a characteristic peripheral nephropathy in humans and animals; the chronic MRL is based on this effect in humans. Commercial hexane, which contains *n*-hexane plus other C_6 branched chain and cyclic alkanes (see Table 6-8), also has been shown to cause this effect in animals, due to its content of *n*-hexane (IRDC 1981) (see Section 6.2.4.1). The non *n*-hexane portion of the mixture does not. In addition, the non *n*-hexane constituents of this combined fraction do not appear to cause peripheral neuropathy when tested singly although, like *n*-hexane, they do cause neurological effects (depression of the central nervous system). *n*-Hexane and commercial hexane are respiratory irritants. Commercial hexane has undergone extensive recent testing as part of an EPA Test Rule under TSCA Section 4. However, until the database for commercial hexane can be more fully evaluated, the chronic MRL for *n*-hexane has been determined to be the most appropriate surrogate for a health guidance value for this fraction.

Oral Exposure. Health effects data regarding oral exposure to this fraction are limited and available mainly for *n*-hexane. *n*-Hexane caused peripheral neuropathy in two species of animals, indicating that effects by the oral route may be similar to those by the inhalation route. ATSDR concluded that the incompleteness of the oral database precluded derivation of oral MRLs for this compound.

Aliphatic $EC_{>8}$-EC_{16} Combined Fraction. The combined fraction consists of the following three fractions:

$EC_{>8}$- EC_{10}: *n*-nonane, *n*-decane, branched chain C_9C_{10} alkanes, substituted cycloalkanes, a few alkenes

Table 6-8. Inhalation MRLs, Critical Effects, and EPA Cancer Assessments for Aliphatic EC_5–EC_8 Fraction

C	EC	Compound	ATSDR Toxicological Profile	Acute		Intermediate		Chronic		
				MRL		MRL		MRL		EPA Cancer WOE, risk per ppm[a]
				ppm	Effect	ppm	Effect	ppm	Effect[a]	
EC_5-EC_6										
6	6	*n*-Hexane	1999b	–	–	–	–	0.6	Neurological	D, NA[b]
6	5.68–6.59	Commercial hexane[c]	(1999b)[c]	–	–	–	–	–	[Respiratory, reproductive?][c]	–[d]
$EC_{>6}$-EC_8										
7	7	n-heptane	–	–	–	–	–	–	–	D, NA
7	7.22	Methylcyclohexane	–	–	–	–	–	–	[Renal?][e]	D, NA[e]
8	6.89	2,2,4-Trimethyl-pentane	–	–	–	–	–	NV	–	–

[a] EPA cancer WOE and risk are from the cited ATSDR toxicological profile and/or IRIS (EPA 1998b), unless otherwise specified. Critical effects are listed in brackets for mixtures or compounds that are not the subjects of ATSDR toxicological profiles, but have been evaluated by other agencies for the purposes of deriving health effects criteria. These health effects are shown only to indicate potentially sensitive endpoints of exposure to that fraction, and do not have the same level of confidence as an ATSDR assessment.

[b] The WOE was determined by EPA (1989a).

[c] A mixture of C_6 alkanes, including ≈50% *n*-hexane. ATSDR (1999b) presented some toxicological data on commercial hexane, but did not consider the derivation of MRLs for this mixture. The TPHCWG (1997b) concluded that the above-listed effects were the critical effects, based on abstracts of unpublished 2-year inhalation studies of commercial hexane in rats and mice (Daughtrey et al 1994; Kelly et al 1994), which were not cited by ATSDR (1999b). The commercial hexane contained 53% n-hexane, 16% 3-methylpentane, 14% methylcyclopentane, 12% 2-methylpentane, 3 % cyclohexane, 1% 2,3-dimethylbutane, and <1% other constituents. The NOAELs chosen by the TPHCWG as the basis for the RfCs appear to be higher than the LOAELs for maternal toxicity in mice and rats in inhalation developmental toxicity studies (Bushy Run 1989a, 1989b). In addition, unpublished 26-week inhalation studies of a mixture of C_6 hexanes of approximately the same composition reported histopathologic evidence of peripheral neuropathy at a duration-adjusted LOAEL lower than the LOAELs in the 2-year studies (IRDC 1981). Thus, the conclusions of the TPHCWG regarding the critical effect need additional evaluation.

[d] Abstracts of unpublished chronic inhalation carcinogenicity studies of commercial hexane in rats and mice report evidence of carcinogenicity in female mice (Daughtrey et al. 1994; Kelly et al. 1994).

[e] Although EPA (1989c, 1997a) concluded that the critical effect of inhalation exposure methylcyclohexane was renal effects in male rats in a 1-year inhalation study with a postexposure observation period (Kinkead et al 1985), the effects appear to have been associated with $\alpha_{2\mu}$-globulin nephropathy, and thus may not be relevant to human health. The WOE was determined by EPA (1989c).

C = carbon number; EC = Equivalent Carbon Number Index; MRL = minimal risk level; NA = not applicable; NV = not verifiable; the health effects data for this compound were reviewed by the EPA RfD/RfC Work Group and determined to be inadequate for the derivation of an RfC (EPA 1998b); WOE = weight-of-evidence classification for carcinogenicity

$EC_{>10}$- EC_{12} and $EC_{>12}$- EC_{16}: longer chain *n*-alkanes; probably larger branched and cyclic alkanes, but EC values not provided (TPHCWG 1997a).

Inhalation Exposure. Health effects data are available for inhalation exposure to some petroleum products corresponding to this combined fraction. Intermediate MRLs of 3 mg/m^3 for JP-5 and JP-8 and 0.01 mg/m^3 for kerosene, and a chronic MRL of 0.3 mg/m^3 for JP-7 have been derived; these are listed in Table 6-9. These four fuels are similar in composition, consisting primarily of aliphatics in the C_9-C_{16} range. All contain some significant aromatic components. In addition, health effects data from studies of two dearomatized petroleum streams have been evaluated by the TPHCWG (1997c). The sensitive effect for exposure to all these products is hepatic. The effect for kerosene, however, was a decrease in blood glucose levels, attributed to hepatic effects. The MRL for kerosene, based on this effect, appears to involve greater uncertainty as to the toxicological significance of the effect. As a result, the intermediate MRL of 3 mg/m^3 and chronic MRL of 0.3 mg/m^3 for the jet fuels have been determined to be the most appropriate surrogate values for the assessment of health effects due to exposure to this fraction.

Oral Exposure. Limited data are available for health effects of oral exposure to this combined fraction. Three studies of dearomatized petroleum streams have been evaluated by the TPHCWG (1997c) for use in RfD derivations, but these studies are unpublished and unreferenced. In addition, a study of JP-8 (Mattie et al. 1995) was used for RfD derivation by the TPHCWG (1997c). The critical effects are listed in Table 6-10. There are no MRLs relevant to this fraction. The sensitive effect of the dearomatized streams was hepatic. Some slight indications of hepatic effects were also seen in the study of JP-8, but no histopathological effects or changes in absolute organ weight.

Aliphatic $EC_{>16}$- EC_{35} Combined Fraction. The combined fraction consists of the following fractions:

$EC_{>16}$- EC_{21}: *n*-hepta-, *n*-octa-, and *n*-nonadecane, *n*-eicosadecane, and probably branched and cyclic alkanes

$EC_{>21}$- EC_{35}: longer chain *n*-alkanes and probably branched and cyclic alkane

Table 6-9. Inhalation MRLs, Critical Effects, and EPA Cancer Assessments for Aliphatic $EC_{>8}$–EC_{16} Fraction

C	EC	Compound or Mixture	ATSDR Toxicological Profile	Acute		Intermediate		Chronic		
				MRL		MRL		MRL		EPA Cancer WOE, risk per mg/m^{3a}
				mg/m^3	Effect	mg/m^3	Effect[a]	mg/m^3	Effect	
10–11	–	C_{10}–C_{11} Iso-paraffinic solvent[b]	–	–	–	–	[Hepatic, adaptive][b]	–	–	–
7–11	–	Dearomatized white spirit[c]	–	–	–	–	[Hepatic, adaptive][c]	–	–	–
≈9–16	–	JP-7	1995c	–	–	–	–	0.3	Hepatic	–
9–16	–	JP-5, JP-8[d]	1998b	–	–	3[d]	Hepatic	–	–	–
9–16	–	Kerosene	1995g	–	–	0.01	Metabolic (hepatic)	–	–	–

[a] EPA cancer WOE and risk are from the cited ATSDR toxicological profile and/or IRIS (EPA 1998b). Critical effects are listed in brackets for mixtures or compounds that are not the subjects of ATSDR toxicological profiles, but have been evaluated by other agencies for the purposes of deriving health effects criteria. These health effects are shown only to indicate potentially sensitive endpoints of exposure to that fraction, and do not have the same level of confidence as an ATSDR assessment.

[b] A mixture composed of C_{10}–C_{11} branched-chain alkanes. According to the TPHCWG (1997b), the listed effect, seen in a 12-week inhalation study in rats (Phillips and Eagan 1984), was adaptive rather than adverse.

[c] A mixture composed of C_7–C_{11} branched, straight, and cyclic alkanes. According to the TPHCWG (1997b), the listed effect, seen in a 12-week inhalation study in rats (Phillips and Eagan 1984), was adaptive rather than adverse.

[d] The intermediate inhalation MRL was derived for JP-5 and JP-8 based on a study of JP-5.

C = carbon number; EC = Equivalent Carbon Number Index; MRL = minimal risk level; NA = not applicable; WOE = weight-of-evidence classification for carcinogenicity

Table 6-10. Oral MRLs, Critical Effects, and EPA Cancer Assessments for Aliphatic $EC_{>8}$–EC_{16} Fraction

C	EC	Compound or mixture	ATSDR Toxicological Profile	Acute		Intermediate		Chronic		
				MRL		MRL		MRL		EPA Cancer WOE, risk per mg/kg/day[a]
				mg/kg/day	Effect	mg/kg/day	Effect[a]	mg/kg/day	Effect	
9–12	–	C_9–C_{12} Dearomatized aliphatic[b]	–	–	–	–	[Hepatic][b]	–	–	–
10–13	–	C_{10}–C_{13} Dearomatized aliphatic[c]	–	–	–	–	[Hepatic][c]	–	–	–
11–17	–	C_{11}–C_{17} Isoparaffinic solvent[d]	–	–	–	–	[Hepatic][d]	–	–	–

[a] EPA cancer WOE and risk are from the cited ATSDR toxicological profile and/or IRIS (EPA 1998b). Critical effects are listed in brackets for mixtures or compounds that are not the subjects of ATSDR toxicological profiles, but have been evaluated by other agencies for the purposes of deriving health effects criteria. These health effects are shown only to indicate potentially sensitive endpoints of exposure to that fraction, and do not have the same level of confidence as an ATSDR assessment.

[b] A mixture composed of C_9–C_{12} branched, straight, and cyclic alkanes. The TPHCWG (1997c) concluded that the listed effect, which occurred in an unpublished and unreferenced 90-day oral study of this mixture in rats, was the critical effect.

[c] A mixture composed of C_{10}–C_{13} branched, cyclic, and straight alkanes. The TPHCWG (1997b) concluded that the listed effect, which occurred in an unpublished and unreferenced 13-week oral study of this mixture in rats, was the critical effect.

[d] A mixture composed of C_{11}–C_{17} branched and cyclic alkanes. The TPHCWG (1997b) concluded that the listed effect, seen in an unpublished and unreferenced 90-day oral study of this mixture in rats, was the critical effect.

C = carbon number; EC = Equivalent Carbon Number Index; MRL = minimal risk level; NA = not applicable; WOE = weight-of-evidence classification for carcinogenicity

Inhalation Exposure. No information was located on the health effects of inhalation exposure to compounds or mixtures of petroleum hydrocarbons that fall within this fraction.

Oral Exposure. No pertinent assessments by ATSDR exist, but studies of mixtures of mineral oil hydrocarbons have been evaluated by the TPHCWG (for use in deriving RfDs for this fraction). Table 6-l 1 summarizes the pertinent information. The critical effect of these mineral oils was judged to be hepatic.

6.7 RELEVANCE TO PUBLIC HEALTH

This profile covers total petroleum hydrocarbons (TPH), which is defined as the measurable amount of petroleum-based hydrocarbon in an environmental medium (Chapter 2). TPH is measured as the total quantity of hydrocarbons without identification of individual constituents. Sources of TPH contamination in the environment range from crude oil, to fuels such as gasoline and kerosene, to solvents, to mineral- based crankcase oil and mineral-based hydraulic fluids. These products contain not only a large number and variety of petroleum hydrocarbons, but also other chemicals that, strictly speaking, are not the subject of this profile, such as non-hydrocarbon additives and contaminants. The TPH issue is further complicated by the number of petroleum-derived hydrocarbons that have been identified-more than 250-and the variability in composition of crude oils and petroleum products (see Section 3.2 and Appendices D and E for details).

Following a spill, leak, or other release of a petroleum product into the environment, changes occur in the location and composition of the released hydrocarbons, as described in Section 5.3. The smaller molecular weight hydrocarbons, which tend to have relatively high vapor pressures and/or water solubilities, tend to volatilize into the air, dissolve into infiltrating rainwater or groundwater and migrate away from the release area, and biodegrade. The larger molecular weight constituents tend to sorb to soil or sediment and remain relatively immobile.

Because TPH is a complex and highly variable mixture, assessment of health impacts depends on several factors, assumptions, and circumstances. Of prime importance is the specific exposure scenario. For example, immediately following a large release of a “lighter” petroleum product (e.g., automotive gasoline), central nervous system depression could occur in people in the immediate vicinity of the spill if

Table 6-11. Oral MRLs, Critical Effects, and EPA Cancer Assessments for Aliphatic $EC_{>16}$–EC_{35} Fraction

C	EC	Compound	ATSDR Toxicological Profile	Acute		Intermediate		Chronic		
				MRL		MRL		MRL		EPA Cancer WOE, risk per mg/kg/day[a]
				mg/kg/day	Effect	mg/kg/day	Effect[a]	mg/kg/day	Effect	
15–37	–	Low MW Mineral oils[b]	–	–	–	–	[Hepatic][b]	–	–	–
27–45	–	High MW Mineral oils[c]	–	–	–	–	[Hepatic][c]	–	–	–

[a] EPA cancer WOE and risk are from the cited ATSDR toxicological profile and/or IRIS (EPA 1998b). Critical effects are listed in brackets for mixtures or compounds that are not the subjects of ATSDR toxicological profiles, but have been evaluated by other agencies for the purposes of deriving health effects criteria. These health effects are shown only to indicate potentially sensitive endpoints of exposure to that fraction, and do not have the same level of confidence as an ATSDR assessment.

[b] Five mixtures of cyclic alkanes with the following carbon ranges (15–30, 17–30, 21–35, and 22–37) and one mixture of branched chain alkanes of carbon range 18–30 were tested in a 90-day oral study in rats (Smith et al. 1996). The TPHCWG (1997b) concluded that the listed effect was the critical effect. The conclusion that one of the observed effects (mesenteric lymph node histiocytosis) was not adverse may need further evaluation, as does the existence of a LOAEL for one of these mixtures (Firriolo et al 1995) at a dose slightly lower than the NOAEL in the selected study.

[c] Two mixtures of branched-chain alkanes with carbon ranges of 27–43 and 28–45 were tested in a 90-day oral study in rats (Smith et al. 1996). The TPHCWG (1997b) concluded that the listed effect was the critical effect. The conclusion that one of the observed effects (mesenteric lymph node histiocytosis) was not adverse may need further evaluation.

C = carbon number; EC = Equivalent Carbon Number Index; MW = Molecular weight; MRL = minimal risk level; NA = not applicable; WOE = weight-of-evidence classification for carcinogenicity

they inhaled the volatilized components. In a confined or poorly ventilated area, asphyxiation would even be a concern. Contamination of groundwater and surface water with the soluble components (e.g., the BTEXs) could impact drinking water sources. Exposure to a contaminated water supply may take place over a period of weeks or years, and raises concerns for more subtle nervous system effects, developmental effects, and cancer. The less volatile or soluble constituents (such as benzo(a)pyrene) may tend to remain in the area of the release for extended periods. Even during the early stages of this release scenario, exposures will tend to be to fractions of the product (the more volatile or more soluble compounds) rather than to the whole product. Therefore, public health assessments for TPH require knowledge of the specific fractions and/or chemicals at the point of exposure (e.g., drinking water well, soil, air). These data are summarized in this toxicological profile (particularly Sections 3.2 and 6.3) and provided in more detail in the toxicological profiles on the individual components and whole products.

A central tool in ATSDR assessment of public health impacts is the minimal risk level (MRL) health guidance value. MRLs have been developed by ATSDR for many hazardous waste constituents, though no new MRLs have been developed for TPH. A limited number of existing MRLs can be applied to TPH assessment. Most are MRLs for individual TPH components (e.g., benzene); however, a few MRLs are available for whole petroleum products. MRLs for substances that represent the fractions defined by the ATSDR approach to assessing TPH health impacts are provided and discussed in this profile. In recognition of the likelihood that even acute exposures to fresh releases will be to fractions of a product, the information on pertinent fractions of TPH should also be consulted (particularly Sections 2.3, 6.1, 6.2 and 6.6).

In the case of weathered releases, the fraction approach is likely to be the most useful. Analytical methods that support the fraction approach should be chosen to characterize exposures (Section 3.3, TPHCWG approach). The identity of the original contaminating product(s) need not be known. Health effects data for these fractions are discussed in Section 6.2 and recommendations for fraction-specific MRLs and for cancer assessment are presented in Section 6.6.

The issue of exposure to complex mixtures was introduced and briefly discussed in Section 6.1.1. In Sections 6.1.2 and 6.1.3 other related TPH approaches are discussed. The ATSDR fraction approach preferentially adopts MRLs for petroleum products that are similar in composition to the transport fraction. When no such data are available, a surrogate MRL from a representative constituent of the

fraction is adopted for the entire mass of the fraction, a practice which implicitly assumes that the toxicity of the constituents of a fraction is additive. This approach is consistent with existing ATSDR and EPA guidance (ATSDR 1992; De Rosa et al. 1996; EPA 1986; Johnson and De Rosa 1995; Mumtaz et al. 1994).

Additional refinements to the fraction approach for assessing health effects include estimation of an index of concern (IOC) for the indicator compounds (the BTEXs) of the aromatic EC_5-EC_9 fraction, or to account for exposure to more than one fraction. This approach is also based on the assumption of additivity, and is reasonable for compounds or fractions that affect the same system or target organ. The IOC is the sum of the ratios of the monitored level of exposure to the accepted level of exposure for each of the constituents of a mixture:

$$IOC = E_1,/AL_1 + E_2AL_{2...} + E_i/AL_i$$

where:

E_1 = the actual exposure level to the ith component
$AL,$ = the acceptable exposure level for the ith component

The accepted levels of exposure for ATSDR assessments would be inhalation MRLs, or soil or water concentrations calculated from oral MRLs. For example, the IOC method could be applied to acute oral exposures to the aromatic EC_5-EC_9 fraction (toluene, *p*-xylene) and the aromatic $EC_{>9}$-EC_{16} fraction, for which the critical effects are neurological (Table 6-2).

Other refinements could be provided by implementing the target-organ toxicity dose approach, which attempts to estimate the plausible critical effect and IOC that would have been calculated had the particular mixture been tested (Mumtaz et al. 1994, 1997). This approach is complicated, and would be suggested only when additional assessment is needed, perhaps to resolve differences between expected and actual health effects outcomes, or where critical effects are different across constituents or fractions that make up the “mixture.”

Another complicated mixtures assessment method under investigation by ATSDR is the weight-of- evidence method for interactions (De Rosa et al. 1996; Johnson and De Rosa 1995; Mumtaz et al. 1994;

Mumtaz and Durkin 1992). This method provides adjustments to the IOC to take into account interactions between the constituents of the mixture. Application to the BTEXs, particularly benzene and toluene, for which interactions have been reasonably well characterized, may be fruitful if needed to resolve issues in a health assessment.

Regardless of the circumstances and methods, TPH health assessments are limited by data gaps in the toxicology for many of the compounds, transport fractions, and mixtures of petroleum products and wastes. The limitations of the analytical method(s) used to generate the TPH data must be understood (e.g., whether the analytical method identified transport fractions or specific compounds) (see Section 3.3). As long as the uncertainties and data limitations are recognized, the method described in Section 6.1.3 and the health effects information in Sections 6.2 and 6.3 provide general guidance for health assessments for TPH.

6.8 BIOMARKERS OF EXPOSURE AND EFFECT

Biomarkers are broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility (NAS/NRC 1989).

Due to a nascent understanding of the use and interpretation of biomarkers, implementation of biomarkers as tools of exposure in the general population is very limited. A biomarker of exposure is a xenobiotic substance or its metabolite(s), or the product of an interaction between a xenobiotic agent and some target molecule(s) or cell(s) that is measured within a compartment of an organism (NAS/NRC 1989). The preferred biomarkers of exposure are generally the substance itself or substance-specific metabolites in readily obtainable body fluid(s) or excreta. However, several factors can confound the use and interpretation of biomarkers of exposure. The body burden of a substance may be the result of exposures from more than one source. The substance being measured may be a metabolite of another xenobiotic substance (e.g., high urinary levels of phenol can result from exposure to several different aromatic compounds). Depending on the properties of the substance (e.g., biologic half-life) and environmental conditions (e.g., duration and route of exposure), the substance and all of its metabolites may have left the body by the time samples can be taken. It may be difficult to identify individuals exposed to hazardous substances that are commonly found in body tissues and fluids (e.g., essential mineral nutrients such as

copper, zinc, and selenium). Biomarkers of exposure to total petroleum hydrocarbons are discussed in Section 6.8.1.

Biomarkers of effect are defined as any measurable biochemical, physiologic, or other alteration within an organism that, depending on magnitude, can be recognized as an established or potential health impairment or disease (NAS/NRC 1989). This definition encompasses biochemical or cellular signals of tissue dysfunction (e.g., increased liver enzyme activity or pathologic changes in female genital epithelial cells), as well as physiologic signs of dysfunction such as increased blood pressure or decreased lung capacity. Note that these markers are not often substance specific. They also may not be directly adverse, but can indicate potential health impairment (e.g., DNA adducts). Biomarkers of effects caused by total petroleum hydrocarbons are discussed in Section 6.8.2.

A biomarker of susceptibility is an indicator of an inherent or acquired limitation of an organism's ability to respond to the challenge of exposure to a specific xenobiotic substance. It can be an intrinsic genetic or other characteristic or a preexisting disease that results in an increase in absorbed dose, a decrease in the biologically effective dose, or a target tissue response. Biomarkers of susceptibility are discussed in Section 6.10, Populations That Are Unusually Susceptible.

More information on biomarkers of exposure and effect to specific petroleum hydrocarbons can be found in ATSDR toxicological profiles on benzene (ATSDR 1997a), toluene (ATSDR 1994), ethylbenzene (ATSDR 1999a), xylenes (ATSDR 1995d), hexane (ATSDR 1999b), naphthalene (ATSDR 1995e) and polycyclic aromatic hydrocarbons (ATSDR 1995f); information for specific petroleum products can be found in ATSDR profiles on automotive gasoline (ATSDR 1995a), fuel oils (ATSDR 1995g), jet fuels (ATSDR 1995c 1998b), mineral-based crankcase oils (ATSDR 1997c), hydraulic fluids (ATSDR 1997b), and Stoddard solvent (ATSDR 1995b).

6.8.1. Biomarkers Used to Identify or Quantify Exposure to TPH

Because of the compositional complexity of TPH, detection of specific hydrocarbons or their metabolites in biological fluids or tissues cannot be expected to provide a reliable biomarker of exposure to petroleum-derived hydrocarbons in general. However, detection of specific hydrocarbons (or their metabolites) from several aromatic and/or aliphatic fractions in biological fluids or tissues can provide reliable evidence of exposure. Examples of proposed biomarkers of exposure to petroleum products include: benzene in

exhaled air and phenol in urine to indicate exposure to gasoline (IARC 1989a), the odor of kerosene on the breath or clothing to indicate oral or dermal exposure to kerosene, and radiological findings of lung infiltrations to indicate oral or inhalation exposure to kerosene or other petroleum products (ATSDR 199.58; Snodgrass 1997). Lipid granulomatas found in autopsied livers and spleens (i.e., lipoid droplets surrounded by lymphocytes and macrophages) are thought to be caused by dietary exposure to mineral oils and waxes (Wanless and Geddie 1985; Miller et al. 1996); their detection in autopsied tissues may be useful as an index of exposure to petroleum hydrocarbons, especially hydrocarbons in the aliphatic $EC_{>16}$-EC_{35} fractions.

6.8.2 Biomarkers Used to Characterize Effects Caused by TPH

Symptoms of neurological dysfunction, such as ataxia, poor coordination and gait irregularities, are potential biomarkers of effect from acute or repeated high-level exposure to petroleum-derived hydrocarbons in the aliphatic EC_5-EC_8 and aromatic EC_5-EC_9 fractions (see ATSDR 1994, 1995a, 1995c, 1995d, 1995f, 1997a, 1998b, 1999a, 1999b). Such symptoms, while shared by many hydrocarbons in these fractions, are not specific to petroleum hydrocarbons and could indicate exposure to other substances such as halogenated hydrocarbons or neurotoxic metals. Such symptoms, however, are not expected from the low-level exposure to hydrocarbons in these fractions that is likely to be experienced by people residing in the vicinity of disposal sites contaminated with petroleum hydrocarbons.

Measurements of motor and sensory nerve conduction velocities and action potential amplitudes have been proposed as sensitive preclinical biomarkers of peripheral neuropathy in workers repeatedly exposed to *n*-hexane (ATSDR 1999b), but this effect is specific to *n*-hexane (and perhaps a few other aliphatic hydrocarbons in the EC_5-EC_8 fraction) among petroleum hydrocarbons.

Many, but not all, PAHs (aromatic $EC_{>16}$-EC_{35} hydrocarbons) are genotoxic in various test systems and carcinogenic in animal test systems. The measurement of benzo(a)pyrene-DNA adducts in human body tissues or fluids has been proposed as a biomarker of effect from exposure to combustion or pyrolytic products containing genotoxic and carcinogenic PAHs, of which benzo(a)pyrene is the most extensively studied (see ATSDR 1995f). These measurements, however, are specific to benzo(a)pyrene and do not identify the source of the benzo(a)pyrene (PAHs are ubiquitous in the environment because they are produced by the pyrolysis or combustion of any material containing hydrocarbons).

Hematological effects from exposure to hydrocarbons in the aromatic EC_5-EC_9 and $EC_{>9}$-EC_{16} fractions include hemolytic anemia from naphthalene exposure and decreased hematopoiesis and leukemia from benzene exposure. Because these effects are not specific to these hydrocarbons, frequent monitoring of blood cell counts in benzene-exposed workers has been used as a biomarker of hematotoxic effects (see ATSDR 1997a).

6.9 INTERACTIONS WITH OTHER SUBSTANCES

Individuals exposed to TPH in the environment are exposed to complex mixtures that are not generally restricted to hydrocarbons alone. It is reasonable to expect that components of such complex mixtures may interact to produce additive effects that do not influence the toxicity of individual components, and synergistic or antagonistic effects that do. Studies with the BTEXs (see ATSDR 1994, 1995d, 1997a, 1999a), with naphthalene and methylnaphthalenes (see ATSDR 1995e), with PAHs (ATSDR 1995f), and with hexane (ATSDR 1999b) indicate that competitive or non-competitive inhibitory interactions with active sites of cytochrome P-450 isozymes, epoxide hydrolases, or other enzymes can influence metabolism of individual hydrocarbons. This can lead to antagonism of toxic effects mediated by metabolic intermediates (e.g., hematopoietic and cancer effects from benzene, cancer, or genotoxic effects from carcinogenic PAHs such as benzo(a)pyrene or dibenz(a,h)anthracene; peripheral neuropathy from hexane) or synergism or potentiation of toxic effects mediated by the parent hydrocarbon (e.g., acute CNS depression from the BTEXs). In addition, inductive or enhancing effects on enzyme activities can increase metabolic rate or capacity leading to potential non-additive interactive effects on hydrocarbon toxicities: potential synergism or potentiation toxic effects with induction of enzymes catalyzing the production of toxic intermediates, and potential antagonism of toxic effects with induction of detoxifying enzymes. Given the compositional complexity of TPH mixtures that may be found in the environment, it is difficult, if not impossible, to make reliable statements predicting the magnitude and direction of specific interactions that may occur. In the face of such large uncertainty, assuming that chemicals in complex mixtures interact in an additive manner at a particular target organ may be the most reasonable approach because it is the most simple.

6.10 POPULATIONS THAT ARE UNUSUALLY SUSCEPTIBLE

A susceptible population will exhibit a different or enhanced response to petroleum hydrocarbons than will most people exposed to the same level of petroleum hydrocarbons in the environment. Reasons may include genetic makeup, age, health and nutritional status, and exposure to other toxic substances (e.g., cigarette smoke). These parameters may result in reduced detoxification or excretion of petroleum hydrocarbons, or compromised function or organs affected by petroleum hydrocarbons.

Factors that inhibit or alter the activity of the mixed function oxidase enzymes may increase the risk from exposure to the indicator compounds in the aromatic EC_5-EC_9 fraction (the BTEXs), the aromatic $EC_{>16}$-EC_{35} fraction (the carcinogenic PAHs in this fraction) and a constituent of the aliphatic EC_5-EC_8 fraction (*n*-hexane). For example, concurrent alcohol consumption may increase the risk of central nervous system depression from the BTEXs, ototoxicity from toluene, and hematotoxicity from benzene. Acetone exposure may increase the risk of peripheral neuropathy of *n*-hexane. People who take haloperidol, acetaminophen, or aspirin, or who have a nutritionally inadequate diet, may also be more susceptible to the toxicity of these agents. ATSDR (1995f) noted that a substantial percentage of children consume less than the recommended dietary allowances of certain nutrients.

Other populations are unusually susceptible to the aromatic EC_5-EC_9 fraction. People with l3-thalassemia may be at risk for benzene exposure because some forms of β-thalassemia may exacerbate the adverse effects of benzene on the hematopoietic system. Children and fetuses may be at increased risk to benzene toxicity because their hematopoietic cell populations are expanding and dividing cells are at a greater risk than quiescent cells. Developmental effects in animals are the basis for intermediate inhalation MRLs for ethylbenzene and mixed xylene, indicating that the embryo/fetus may be particularly sensitive to these two BTEXs. People with subclinical and clinical epilepsy are considered at increased risk of seizures from xylene because of its central nervous system effects.

Person with inherited erythrocyte G6PD deficiency have an enhanced susceptibility to the hemolytic effects of naphthalene, a constituent of the aromatic $EC_{>9}$-EC_{16} fraction. Infants appear to be more sensitive than adults to this effect, and infants are more prone to permanent neurological damage as a consequence of the jaundice that results from the hemolysis. Naphthalene has been shown to cross the human placenta to

cause hemolysis and hemolytic anemia in the newborn infants of mothers who consumed naphthalene during pregnancy (ATSDR 1995e).

People with aryl hydrocarbon hydroxylase (AHH) that is particularly susceptible to induction may be more susceptible to the carcinogenic PAHs found in the aromatic $EC_{>16}$-EC_{35} fraction. Individuals undergoing rapid weight loss that includes loss of body fat are anticipated to be at risk because of the systemic release and activation of PAHs that had been stored in fat. People with genetic diseases that are associated with DNA-repair deficiencies (e.g., xeroderma pigmentosum, ataxia telangiectasia, familial retinoblastoma, Down's syndrome) may be more susceptible to PAH-related malignancy. Individuals who have significant exposure to ultraviolet radiation, as from sunlight, may be at increased risk of developing skin cancer from PAH exposure. The human fetus may also be particularly susceptible to PAH toxicity because of increased permeability of the embryonic/fetal blood-brain barrier and a decreased liver-enzyme conjugating function. Based on studies of benzo(a)pyrene in animals, women may be at increased risk of reproductive dysfunction following exposure to high levels of PAHs.

Individuals with impaired pulmonary function may be more susceptible to the respiratory irritant effects of the volatile petroleum hydrocarbons (primarily the aromatic EC_5-EC_9 and aliphatic EC_5-EC_9 fractions).

Additional information regarding populations unusually susceptible to the aliphatic EC_5-EC_8, $EC_{>8}$-EC_{16}, and $EC_{>16}$-EC_{35}, fractions is limited. Factors that alter the function of mixed function oxidase enzymes may increase the risk of peripheral neuropathy from exposure to *n*-hexane, a constituent of the EC_5-EC_8 fraction. A single animal study indicates that susceptibility to the neuropathic effects of *n*-hexane was more severe in young adults than in weanlings. A single study of kerosene ($EC_{>8}$-EC_{16}) in rats showed that younger animals, and particularly preweanlings, were more susceptible than older rats to the lethality of kerosene, but whether these findings for *n*-hexane and kerosene can be extrapolated to humans is uncertain. Case reports of accidental poisoning through ingestion indicate that children 5 years old or younger often mistakenly drank kerosene because it was accessible. The applicability of this scenario to hazardous waste sites is questionable.

More detailed information regarding populations that are unusually susceptible to petroleum hydrocarbons can be obtained from the ATSDR toxicological profiles (ATSDR 1994, 1995d, 1995e, 1995f, 1997a,

1998b, 1999a, 1999b) on which this section was based. Other pertinent toxicological profiles (ATSDR 1995b, 1995c, 1995g) noted a lack of information on susceptible populations.

6.11 METHODS FOR REDUCING TOXIC EFFECTS

This section will describe clinical practice and research concerning methods for reducing toxic effects of exposure to petroleum hydrocarbons. However, because some of the treatments discussed may be experimental and unproven, this section should not be used as a guide for treatment of exposures to petroleum hydrocarbons. When specific exposures have occurred, poison control centers and medical toxicologists should be consulted for medical advice. The following texts provide specific information about treatment following exposures to petroleum hydrocarbons:

Snodgrass, W.R. 1997. Clinical Toxicology. In: Cassarett and Doull's Toxicology. The Basic Science of Poisons. Fifth Edition. pp. 969-986. C.D. Klaassen, M.O. Amdur, and J. Doull, eds McGraw-Hill, New York.

Friedman, P.A. 1987. Poisoning and Its Management. In: Harrison's Principles of Internal Medicine. Eleventh Edition. pp. 838-850. J.D. Jeffers, E.J. Scott and M. Ramos-Englis, eds. McGraw-Hill. New York.

Klaasen, C.D. 1996. Nonmetallic Environmental Toxicants. Air Pollutants, Solvents and Vapors, and Pesticides. In: Goodman and Gilman's The Pharmacological Basis of Therapeutics. Ninth Edition. J.G. Hardman and L.E. Limbird, eds. McGraw-Hill, New York.

Information on methods that may be effective in reducing absorption, reducing body burdens, or interfering with mechanisms of toxic action of specific petroleum hydrocarbons can be found in ATSDR profiles on the BTEXs (ATSDR 1994, 1995d, 1997a, 1999a), hexane (ATSDR 1999b), naphthalene (ATSDR 1995f), and PAHs (1995f). Additional information for petroleum products can be found in ATSDR profiles on automotive gasoline (ATSDR 1995a), fuel oils (ATSDR 19958) jet fuels (ATSDR 199512, 1998b), mineral-based crankcase oils (ATSDR 1997c) hydraulic fluids (1997b), and Stoddard solvent (ATSDR 1995b).

6.11.1 Reducing Peak Absorption Following Exposure

It is commonly recognized that, in the treatment of poisoning from ingestion of low viscosity, aliphatic or aromatic hydrocarbons found in petroleum products such as gasoline and kerosene, care must be taken to prevent aspiration into the respiratory tract (Friedman 1987; Klaassen 1996; Snodgrass 1997). Emesis, gastric lavage, and treatment with activated charcoal are often avoided unless large amounts have been ingested (>l00 mL) or there is a known risk of absorption of non-hydrocarbon additives (e.g., metals, pesticides) that may produce systemic effects. If gastric lavage is applied, an endotracheal tube with inflatable cuff is often used to prevent aspiration. Viscous, large molecular weight aliphatic hydrocarbons such as those in mineral oil, heavy lubricants, and Vaseline are not aspirated to the lung and have cathartic properties; removal treatments are not usually used. Absorption of petroleum hydrocarbons by the skin following dermal exposure can be reduced by washing with a mild soap or detergent and water, taking care not to abrade the skin.

6.11.2 Reducing Body Burden

Petroleum-derived hydrocarbons and their metabolites (e.g., fatty acids), especially those in the aliphatic and aromatic $EC_{>16}$-EC_{35} fractions, tend to accumulate in the liver, spleen, and adipose tissues. There are no known clinical methods to facilitate or accelerate removal of petroleum hydrocarbons or their metabolites from these tissues.

6.11.3 Interfering with the Mechanism of Action for Toxic Effects

Acute inhalation or aspiration of ingested aliphatic or aromatic petroleum hydrocarbons of low viscosity can lead to pulmonary irritation and hydrocarbon pneumonia, an acute hemorrhagic necrotizing disease. To counteract secondary bacterial infections and pulmonary edema, antibiotics and oxygen therapy are often applied when indicated by symptoms in particular patients (Klaassen 1996; Snodgrass 1997).

Specific aliphatic and aromatic hydrocarbons found in petroleum products are known to be-metabolized via cytochrome P-450 pathways to reactive metabolic intermediates that are thought to cause non-cancer and cancer effects from chronic exposure (e.g., peripheral neuropathy from 2,5-hexadione, a metabolite of hexane, and cancer effects from various intermediary metabolites of benzene and carcinogenic PAHs). There are no known clinical methods to interfere with these mechanisms of action. However, current research programs are studying the basis of how the consumption of cruciferous vegetables may protect

against chemical carcinogenesis, and examining the protective role that may be played by dietary antioxidants and the induction of Phase II enzymes (enzymes involved in the detoxification of products of cytochrome P-450 enzymes) (see Prochaska and Talalay 1992; Zhang et al. 1992; Talalay 1992; Fahey et al. 1997). Results from this type of research may lead to clinical methods counteracting the toxic effects of chronic exposure to bioactivated hydrocarbons.

6.12 ADEQUACY OF THE DATABASE

The adequacy of the database for many of the constituents of TPH and for petroleum products has been fully discussed in the corresponding toxicological profiles. This section will briefly discuss adequacy of the database to support a fraction-based assessment of TPH.

The database for the aromatic EC_5-EC_9 fraction is that for the individual BTEXs; the recommendation in this profile is to assess each of these compounds individually as indicator compounds. The database for inhalation exposure is more adequate than for oral exposure. Details are provided in the respective ATSDR profiles (ATSDR 1994, 1995d, 1997a, 1999a).

The database for the aromatic $EC_{>9}$-EC_{16} fraction lacks information on a mixture or mixtures that could represent the entire combined fraction. Limited inhalation data are available on a mixture of C9 aromatics (high flash aromatic naphtha, primarily $EC_{9.47}$-$EC_{9.84}$). Health effects data from these mixtures and from potential representative chemicals, including naphthalene, suggest some commonality of effect among constituents of this fraction. MRLs are available for chronic inhalation exposure and all three periods of oral exposure. Surrogate MRL values are suggested for chronic inhalation exposure and acute and intermediate oral exposure to this fraction. Nevertheless, the data do not *strongly* support a surrogate approach. Additional information on the database for naphthalene, 1- and 2-methyl naphthalene, acenaphthylene and acenaphthene is discussed in ATSDR (1995e, 1995f).

The adequacy of the database for the aromatic $EC_{>16}$-EC_{35} fraction, which consists of PAHs, is discussed in ATSDR (1995f). Data for suitable mixtures were not identified. Inhalation data for the individual constituents were particularly limited; no MRLs were available. The oral data support the selection of a surrogate MRL for intermediate exposure to the noncarcinogenic constituents of this fraction, but it is uncertain whether this value is appropriate to represent the noncancer effects of the carcinogenic PAHs.

The database for inhalation exposure to the aliphatic EC_5-EC_8 fraction includes data for a representative mixture, commercial hexane, but many of the studies were performed under a TSCA test rule and have been published only as abstracts (TPHCWG 1997c). ATSDR (1999b) briefly discussed commercial hexane in the toxicological profile on *n*-hexane, but did not consider MRL derivation for commercial hexane, as it was not the subject of the profile. The only compound or petroleum product corresponding to this fraction that has been the focus of MRL derivation by ATSDR is *n*-hexane, for which a chronic inhalation MRL is available. The data were considered inadequate for the derivation of oral MRLs for this compound (ATSDR 1999b). Details of the adequacy of the database for *n*-hexane are provided by ATSDR (1999b).

For the aliphatic $EC_{>8}$-EC_{16} fraction, the database includes a number of studies of petroleum products whose major constituents fall within the EC range of this fraction. These included dearomatized petroleum streams and fuels (JP-5, JP-7, JP-8, kerosene). Studies of the dearomatized petroleum streams are largely unpublished, include oral studies in animals, and have been reviewed by the TPHCWG (1997c). The critical effects were judged to be hepatic. MRLs were available for intermediate and chronic inhalation exposure to JP-7 and JP-5 and JP-8; these are based on hepatic effects. The MRLs for these jet fuels appeared suitable to represent the health effects of the fraction. Detailed analyses of the adequacy of the database for the fuels are provided by ATSDR (1995c, 1995g), 1998).

Mineral oils, which are petroleum products similar in composition to the aliphatic $EC_{>16}$-EC_{35} fraction, have been tested by the oral route, as reviewed by the TPHCWG (1997c); the TPHCWG based its derivation of health effects criteria on these studies. Issues regarding the TPHCWG's derivation include the classification of histiocytosis as a nonadverse effect and the suitability of the F344 rat to serve as a model for humans for this class of compounds (Section 6.2.6.2). ATSDR has not considered the health effects of these products in a toxicological profile, and there are no other petroleum products or constituents corresponding to this fraction that have MRLs.

Ongoing studies of interest are the studies performed under a Section 4 TSCA test rule of commercial hexane and of cyclohexane mentioned by the TPHCWG (1997c). In addition, the Verhaar et al. (1997) describe a proposed approach and ongoing research to develop PBPK/PD models for use in assessing human health risks from exposure to JP-5.

7. REGULATIONS AND ADVISORIES

The international, national, and state regulations and guidelines regarding total petroleum hydrocarbons (TPH) in air, water, and other media are summarized in Table 7-1. No health or environmental benchmarks have been developed for TPH as a general category, though many exist for individual petroleum chemicals or products, such as gasoline.

Benzene is on the list of chemicals in "The Emergency Planning and Community Right-to-Know Act of 1986" (EPA 1988c, 1989c, 1989d). Section 313 of Title III of the Superfund Amendments and Reauthorization Act (SARA) requires owners and operators of certain facilities that manufacture, import, process, or otherwise use the chemicals on this list to report annually any release of those chemicals to any environmental media over a specified threshold level.

OSHA requires employers of workers who are occupationally exposed to petroleum distillates to institute engineering controls and work practices to reduce and maintain employee exposure at or below permissible exposure limits (PEL). The PEL for petroleum distillates is 500 ppm (OSHA 1974).

TPH as oil is regulated by the Clean Water Act as stated in Title 40, Sections 109-l 14 and Section 112 of the Code of Federal Regulations. Sections 109-l14 address oil pollution prevention and spill response. Section 112 pertains to stormwater discharge permitting under the National Pollutant Discharge Elimination System. Underground injection control is regulated according to 40 CFR Sections 144 and 146.

Under Subtitle C of the Resource Conservation and Recovery Act (RCRA), certain wastes containing designated TPH compounds and petroleum-related industrial wastes are listed as hazardous. However, RCRA excludes some TPH-related wastes from regulations (e.g., certain exploration, well development, and productions wastes). The RCRA-listed wastes are also controlled under the Comprehensive Environmental, Response, Compensation, and Liability Act (CERCLA) for accidental releases to the environment.

The American Society for Testing and Materials (ASTM) developed a guide for the community of engineering firms, environmental and risk assessment scientists, and governmental agencies to deal with

petroleum contaminated sites. In 1995 ASTM published its *Standard Guide for Risk-Based Corrective Action Applied at Petroleum Release Sites* partly in response to Subtitle I of the Resource Conservation and Recovery Act (RCRA) (ASTM 1995). RCRA directed the U.S. Environmental Protection Agency (EPA) establish programs to prevent, detect, and clean up releases from underground storage tank systems (UST). ASTM's risk-based corrective action (RBCA) is a widely used, decision-making process for the assessment and response to chemical releases, with particular emphasis on petroleum release, based on the protection of human health and the environment. RBCA integrates site assessment, remedial action selection, and monitoring with risk and exposure assessment practices suggested by the EPA. The RBCA process is implemented in a tiered approach that involves increasingly sophisticated levels of data collection and analysis. Site assessment is followed by site classification whereby sites are classified by the urgency of initial response action based on information collected during the site assessment. Section 5.3.3.3, Transport Models, in Chapter 5 presents a brief overview of the tiered RBCA approach and also provides the basic flow chart of the RBCA approach, Figure 5-2.

Table 7-1. Regulations and Guidelines Applicable to Total Petroleum Hydrocarbons

Agency	Description	Information[a]	References
INTERNATIONAL			
WHO	NA		
International Convention for Prevention of Pollution from Ships (MARPOL)	1978 Protocol: Annexes I-V-Oil, Noxious Liquids, etc	Yes	MARPOL 1978
NATIONAL			
Regulations: a. Water:			
EPA OW	Oil Pollution Prevention (spill prevention control and counter-measure planning)	Yes	40 CFR 112 EPA 1973a
	Criteria for State, Local and Regional Oil Removal Contingency Plans	Yes	40 CFR 109 EPA 1971
	Discharge of Oil	Yes	40 CFR 110 EPA 1987
	Liability Limits for Small Onshore Storage Facilities	Yes	40 CFR 113 EPA 1973b
	Civil Penalties for Violation of Oil Pollution Prevention Regulations	Yes	40 CFR 114 EPA 1974
	National Pollutant Discharge Elimination Permit Application: General Permits	Yes	40 CFR 122.28 EPA 1983a
	Stormwater Discharges	Yes	40 CFR 122.26 EPA 1990a
	Procedures for [Permit] Decision-making: Fact sheet for Stormwater Discharge Associated with Industrial Activities (Notice: e.g., asphalt paving and roofing, oil and gas exaction, hazardous waste TSDFs, landfills/application sites)	Yes	58 FR 61146 EPA 1993
EPA-ODW	Underground Injection Control Criteria and Standards for Program: Criteria and Standards for Class II Wells (oil and gas-related)		40 CFR 144 EPA 1983b
	Underground Injection Control Criteria and Standards for Program: Criteria and Standards for Class II Wells (oil and gas-related)	Yes	40 CFR 146, Subpart C EPA 1980a
Bureau of Land Mgt.	Onshore Oil and Gas Operations: Environment and Safety		43 CFR 3160

Table 7-1. Regulations and Guidelines Applicable to Total Petroleum Hydrocarbons (continued)

Agency	Description	Information[a]	References
NATIONAL (cont.)			
b. Other			
DOT	Table of Hazardous Materials and Special Provisions: Gasoline, Petroleum, Crude Oil, Petroleum Distillates, n.o.s., Petroleum Ether, Petroleum Gases, Petroleum Naphtha, Petroleum Oil, Petroleum Spirit, Hydro-carbon Gases	Yes	49 CFR 172.101 DOT 1990
EPA OPPT	PCB Manufacturing, Processing, Distribution in Commerce and Use Prohibition - Disposal Requirements: Incineration	≥50 ppm	40 CFR 761.60 EPA 1979b
EPA OSW	Criteria for Municipal Solid Waste Landfills	Yes	40 CFR 258 EPA 1991a
	Definition of Used Oil	Yes	40 CFR 260.10 EPA 1980b
	Definition of Solid Waste	Yes	40 CFR 261.2 EPA 1985a
	Identification and Listing of Hazardous Waste: Definition of Hazardous Waste: Rebuttable Presumption of Used Oil Total Halogens Deeming Oil Hazardous	Yes	40 CFR 261.3 EPA 1998c
	Exclusions Drilling Fluids, Produced Waters, Etc., Associated with Exploration, Development, or Production	Yes	40 CFR 261.4(b) EPA 1980c
	Petroleum-contaminated Media and Debris Failing Toxicity Characteristic and Subject to Corrective Action	Yes	
	Non-terne plated Used Oil Filters	Yes	
	Requirements for Recyclable Materials - Exclusions (see 40 CFR 266)	Yes	40 CFR 261.6 EPA 1985b
	Toxicity Characteristic Benzene Lead	 0.5 mg/L (0.5 ppm) 5.0 mg/L (5.0 ppm)	40 CFR 261.6 EPA 1990b

Table 7-1. Regulations and Guidelines Applicable to Total Petroleum Hydrocarbons (continued)

Agency	Description	Information[a]	References
NATIONAL (cont.)			
	Hazardous Wastes from Non-specific Sources: Petroleum Refinery Primary and Secondary Oil/Water/Solids Separation Sludges (F037, F038)	Yes	40 CFR 261.31 EPA 1981a
	Hazardous Wastes from Specific Sources: Petroleum Refining Wastes		
	K048 - K052	Yes	40 CFR 261.32 EPA 1981b
	K170 - K172 (proposed)	Yes	60 FR 57747 EPA 1995b
	Standards for the Management of Specific Hazardous Wastes and Specific Types of Hazardous Waste Management Facilities: Hazardous Waste Burned in Boilers and Industrial Furnaces		40 CFR 266.104
	Destruction and Removal Efficiency for All Organic Hazardous Constituents	99.99%	EPA 1991a
	Low Risk Waste Exemption	50% of fuel is fossil fuel	40 CFR 266.109 EPA 1991a
	Land Disposal Restrictions: Treatment Standards (numerous constituents)		
	F037 - F038	Yes	40 CFR 268.40 EPA 1988c
	K048 - K052	Yes	
	K170 - K172 (proposed)	Yes	40 FR 57747 EPA 1995b
	Standards for the Management of Used Oil	Yes	57 FR 41566 EPA 1992b
	Underground Storage Tank Standards	Yes	40 CFR 280 EPA 1988b
	Release Response and Corrective Action for US Systems Containing Petroleum or Hazardous Substances	Yes	
EPA OSWER	Designation, Reportable Quantities and Notification		
	F037 - F038	1 lb. each	40 CFR 302.4 EPA 1985c
	K048 - K052	10 lb. each	
	K170 - K172 (proposed)	100 lb. each	60 FR 57747 EPA 1995b

Table 7-1. Regulations and Guidelines Applicable to Total Petroleum Hydrocarbons (continued)

Agency	Description	Information[a]	References
NATIONAL (cont.)			
OSHA	Limits for Air Contaminants - Petroleum Distillates	2,000 mg/m^3 (500 ppm)	29 CFR 1910.1000 OSHA 1974
Guidelines			ACGIH 1994
a. Air:			
ACGIH	Threshold Limit Values	890 mg/m^3	
	Gasoline	(300 ppm)	
	TWA	1,480 mg/m^3 (500 ppm)	
	STEL		
NIOSH	Gasoline -		NIOSH 1992
	LOQ	15 ppm	
	CA		
b. Other:			
EPA	RfC (inhalation)		IRIS 1998b
	Ethylbenzene	1 mg/m^3 (0.2303 ppm)	
	Cumene	0.4 mg/m^3 (0.1134 ppm)	
	Naphthalene	0.003 mg/m^3 (0.00069 ppm)	
	n-Hexane	0.2 mg/m^3 (0.0567 ppm)	
	Toluene	0.4 mg/m^3 (0.1062 ppm)	
	RfD (oral)		
	Cumene	0.1 mg/kg/day	
	n-Hexane	0.06 mg/kg/day	
	Naphthalene	0.02 mg/kg/day	
	Ethylbenzene	0.1 mg/kg/day	
	Anthracene	0.3 mg/kg/day	
	Ancenaphthene	0.06 mg/kg/day	
	Fluoranthene	0.04 mg/kg/day	
	Fluorene	0.04 mg/kg/day	
	Pyrene	0.03 mg/kg/day	
	Toluene	0.02 mg/kg/day	
	Xylene	2 mg/kg/day	
STATE			
Regulations and Guidelines: a. Air:	Average Acceptable Ambient Air Concentrations		NATICH 1992
	Diesel Fuel Emissions		
	30 min.	90 µg/m^3	
TX	Annual	9 µg/m^3	
	Gasoline		
CT	8 hours	1.8x10^4 µg/m^3	
FL-Ft Ldle	8 hours	9 mg/m^3 (9x10^3 µg/m^3)	
FL-Pinella	8 hours	9x10^3 µg/m^3	
	24 hours	2.16x10^3 µg/m^3	
KS	1 year	1.33 µg/m^3	
KS-KC	Annual	1.33 µg/m^3	
MI	Annual	1.3 µg/m^3	
ND	8 hours	8.9 mg/m^3 (8.9x10^3 µg/m^3)	
	1 hour	14.8 mg/m^3 (14.8x10^3 µg/m^3)	

Table 7-1. Regulations and Guidelines Applicable to Total Petroleum Hydrocarbons (continued)

Agency	Description	Information[a]	References
STATE (cont.)			
NV	8 hours	21.4 mg/m^3 (21.4x10^3 µg/m^3)	
OK	24 hours	8.9x10^4 µg/m^3	
TX	30 min. Annual	8.9x10^3 µg/m^3 8.9x10^2 µg/m^3	
VA	24 hours	1.5x10^4 µg/m^3	
	Naphtha		
AZ	24 hours	2.6x10^3 µg/m^3	
CT	8 hours 8 hours	2.7x10^4 µg/m^3 60 µg/m^3	
FL-Pinella	8 hours 24 hours	4x10^3 µg/m^3 9.6x10^2 µg/m^3	
TX	30 min. Annual	4x10^3 µg/m^3 4x10^2 µg/m^3	
VA	24 hours	2.25x10^2 µg/m^3	
	Petroleum Distillates		
FL-Ft Ldle	6 hours	9 mg/m^3 (9x10^3 µg/m^3)	
NY	1 year	3x10^{-2} µg/m^3	
TX	30 min. Annual 30 min. Annual	8.9x10^3 µg/m^3 8.9x10^2 µg/m^3 3.5x10^3 µg/m^3 3.5x10^2 µg/m^3	
	Petroleum Gases, Liquified		
ND	8 hours	18 mg/m^3 (18x10^3 µg/m^3)	
TX	30 min. Annual	1.8x10^4 µg/m^3 1.8x10^3 µg/m^3	
VA	24 hours	3x10^4 µg/m^3	
WA-SWEST	24 hours	5.99x10^3 µg/m^3	
b. Water:	State Administered Underground Injection Control Programs		40 CFR 147 EPA 1984
AL, AK, CO, FL, ID, IL, KS, KY, MD, MI, MS, MT, NE, NV, NM, NY, ND, OH, OK, PA, RI, SD, TN, TX, U, WA, WY	Class II		
AL, AR, CA, CO, CT, DE, FL, GA, ID, IL, KS, LA, ME, MD, MA, MN, NC, ND, OH, OK, OR, RI, SC, SD, TX, U, VT, WA, WV, WI, WY	Indian Lands		

Table 7-1. Regulations and Guidelines Applicable to Total Petroleum Hydrocarbons (continued)

Agency	Description	Information[a]	References
STATE (cont.)			
ME	Drinking Water (guideline)	50 $\mu g/m^3$	FSTRAC 1990
MA	Upper Concentration Limits in Groundwater	100,000 $\mu g/L$ (ppb)	BNA 1999
MA	Upper Concentration Limits in Soil	10,000 $\mu g/g$ (ppm)	BNA 1999
CT	Groundwater Protection Criteria	500 $\mu g/L$ (ppb)	BNA 1999
AK, DE, HI, IN, MA, MD, ME, MN, MO, MS, MT, ND, NE, OK, SD, TN, UT, VA, WA, WI, WV, WY	Groundwater Cleanup Standards[b]	States with TPH Parameter[c]	Judge et al. 1998
AK, AL, AZ, CA, DE, FL, HI, IN, IO, KS, MA, MD, ME, MN, MO, MS, MT, NC, ND, NE, NH, NM, NV, OH, OK, OR, RI, SC, SD, TN, UT, VA, VT, WA, WI, WY	Soil Cleanup Standards[D]	States with TPH Parameter[c]	Judge et al. 1997

[a] "Yes" indicates that a specific value was not appropriate but that the referenced regulation or guideline is applicable.

[b] There are many limitations to presenting these standards in a summary table. Each state should be contacted for complete information. See Judge et al. 1998.

[c] Includes TRPH parameter (total recoverable petroleum hydrocarbons), TEH (total extractable hydrocarbons), gasoline range organics (GRO), and diesel range organics (DRO).

[d] See note "b" above and Judge et al. 1997.

ACGIH = American Conference of Governmental Industrial Hygienists; BNA = The Bureau of National Affairs; CFR = Code of Federal Regulations; DOT = Department of Transportation; EPA = Environmental Protection Agency; LOQ = Limit of Quantitation; NA = not applicable; NATICH = National Air Toxics Information Clearinghouse; NIOSH = National Institute of Occupational Safety and Health; ODW = Office of Drinking Water; OSHA = Occupational Safety and Health Administration; OSW = Office of Solid Wastes; OW = Office of Water; STEL = Short-term Exposure Limit; TLV= Threshold Limit Value; TTO = Total Toxic Organic; TWA = Time-weighted Average; WHO = World Health Organization

8. REFERENCES

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9. GLOSSARY

Absorption-The taking up of liquids by solids, or of gases by solids or liquids.

Acute (Exposure)- Exposure to a chemical for a duration of 14 days or less, as specified in the Toxicological Profiles.

Adsorption-The adhesion in an extremely thin layer of molecules (as of gases, solutes, or liquids) to the surfaces of solid bodies or liquids with which they are in contact.

Adsorption Coefficient (K_{oc})-The ratio of the amount of a chemical adsorbed per unit weight of organic carbon in the soil or sediment to the concentration of the chemical in solution at equilibrium.

Adsorption Ratio (Kd)-The amount of a chemical adsorbed by a sediment or soil (i.e., the solid phase) divided by the amount of chemical in the solution phase, which is in equilibrium with the solid phase, at a fixed solid/solution ratio. It is generally expressed in micrograms of chemical sorbed per gram of soil or sediment.

Benchmark Dose (BMD)-is usually defined as the lower confidence limit on the dose that produces a specified magnitude of changes in a specified adverse response. For example, a BMD_{10} would be the dose at the 95% lower confidence limit on a 10% response, and the benchmark response (BMR) would be 10%. The BMD is determined by modeling the dose response curve in the region of the dose response relationship where biologically observable data are feasible.

Benchmark Dose Model-is a statistical dose-response model applied to either experimental toxicological or epidemiological data to calculate a BMD.

Bioconcentration Factor (BCF)-The quotient of the concentration of a chemical in aquatic organisms at a specific time or during a discrete time period of exposure divided by the concentration in the surrounding water at the same time or during the same period.

Biomarkers-are broadly defined as indicators signaling events in biologic systems or samples. They have been classified as markers of exposure, markers of effect, and markers of susceptibility.

Cancer Effect Level (CEL)-The lowest dose of chemical in a study, or group of studies, that produces significant increases in the incidence of cancer (or tumors) between the exposed population and its appropriate control.

Carcinogen-A chemical capable of inducing cancer.

Case-Control Study-A type of epidemiological study which examines the relationship between a particular outcome (disease or condition) and a variety of potential causative agents (such as toxic chemicals). In a case-controlled study, a group of people with a specified and well-defined outcome is identified and compared to a similar group of people without outcome.

Case Report-describes a single individual with a particular disease or exposure. These may suggest some potential topics for scientific research but are not actual research studies.

Case Series-describes the experience of a small number of individuals with the same disease or exposure. These may suggest potential topics for scientific research but are not actual research studies.

Ceiling Value-A concentration of a substance that should not be exceeded, even instantaneously.

Chronic Exposure-Exposure to a chemical for 365 days or more, as specified in the Toxicological Profiles.

Cohort Study-A type of epidemiological study of a specific group or groups of people who have had a corm-non insult (e.g., exposure to an agent suspected of causing disease or a common disease) and are followed forward from exposure to outcome. At least one exposed group is compared to one unexposed group.

Cross-sectional Study-A type of epidemiological study of a group or groups which examines the relationship between exposure and outcome to a chemical or to chemicals at one point in time.

Data Needs-substance-specific informational needs that if met would reduce the uncertainties of human health assessment.

Developmental Toxicity-The occurrence of adverse effects on the developing organism that may result from exposure to a chemical prior to conception (either parent), during prenatal development, or postnatally to the time of sexual maturation. Adverse developmental effects may be detected at any point in the life span of the organism.

Dose-Response Relationship-the quantitative relationship between the amount of exposure to a toxicant and the incidence of the adverse effects.

Embryotoxicity and Fetotoxicity-Any toxic effect on the conceptus as a result of prenatal exposure to a chemical; the distinguishing feature between the two terms is the stage of development during which the insult occurs. The terms, as used here, include malformations and variations, altered growth, and in utero death.

Environmental Protection Agency (EPA) Health Advisory-An estimate of acceptable drinking water levels for a chemical substance based on health effects information. A health advisory is not a legally enforceable federal standard, but serves as technical guidance to assist federal, state, and local officials.

Epidemiology-refers to the investigation of factors that determine the frequency and distribution of disease or other health-related conditions within a defined human population during a specified period.

Equivalent Carbon Number Index-an index based on the boiling point of a chemical normalized to the boiling point of *n*-alkanes or its retention time in a boiling point gas chromatographic column (GC).

Genotoxicity-a specific adverse effect on the genome of living cells that, upon the duplication of affected cells, can be expressed as a mutagenic, clastogenic or carcinogenic event because of specific alteration of the molecular structure of the genome.

Half-life-a measure of rate for the time required to eliminate one half of a quantity of a chemical from the body or environmental media.

Human Equivalent Concentration--the test concentration from an animal study adjusted for continuous exposure and dosimetric differences in humans and the test animal.

Immediately Dangerous to Life or Health (IDLH)-The maximum environmental concentration of a contaminant from which one could escape within 30 minutes without any escape-impairing symptoms or irreversible health effects.

Incidence-The ratio of individuals in a population who develop a specified condition to the total number of individuals in that population who could have developed that condition in a specified time period.

Index of Concern-the ratio of exposure to the minimal risk level (MRL). A value greater than one indicates that there may be some concern for potential noncancer effects.

Intermediate Exposure-Exposure to a chemical for a duration of 15-364 days, as specified in the Toxicological Profiles.

Immunological Effects-are functional changes in the immune response.

Immunologic Toxicity-The occurrence of adverse effects on the immune system that may result from exposure to environmental agents such as chemicals.

In Vitro-Isolated from the living organism and artificially maintained, as in a test tube.

In Vivo-Occurring within the living organism.

Lethal Concentration$_{(LO)}$ (LC_{LO})-The lowest concentration of a chemical in air which has been reported to have caused death in humans or animals.

Lethal Concentration$_{(50)}$ (LC_{50})-A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population.

Lethal Dose$_{(LO)}$ (LD_{LO})-The lowest dose of a chemical introduced by a route other than inhalation that has been reported to have caused death in humans or animals.

Lethal Dose$_{(50)}$(LD_{50})-The dose of a chemical which has been calculated to cause death in 50% of a defined experimental animal population.

Lethal Time$_{(50)}$ (LT_{50})-A calculated period of time within which a specific concentration of a chemical is expected to cause death in 50% of a defined experimental animal population.

Lowest-Observed-Adverse-Effect Level (LOAEL)-The lowest exposure level of chemical in a study, or group of studies, that produces statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control.

Lymphoreticular Effects-represent morphological effects involving lymphatic tissues such as the lymph nodes, spleen, and thymus.

Malformations-Permanent structural changes that may adversely affect survival, development, or function.

Minimal Risk Level (MRL) -An estimate of daily human exposure to a hazardous substance that is likely to be without an appreciable risk of adverse noncancer health effects over a specified route and duration of exposure.

Modifying Factor (MF)-A value (greater than zero) that is applied to the derivation of a minimal risk level (MRL) to reflect additional concerns about the database that are not covered by the uncertainty factors. The default value for a MF is 1.

Morbidity-State of being diseased; morbidity rate is the incidence or prevalence of disease in a specific population.

Mortality-Death; mortality rate is a measure of the number of deaths in a population during a specified interval of time.

Mutagen-A substance that causes mutations. A mutation is a change in the DNA sequence of a cell's DNA. Mutations can lead to birth defects, miscarriages, or cancer.

Necropsy-The gross examination of the organs and tissues of a dead body to determine the cause of death or pathological conditions.

Neurotoxicity-The occurrence of adverse effects on the nervous system following exposure to a chemical.

No-Observed-Adverse-Effect Level (NOAEL)-The dose of a chemical at which there were no statistically or biologically significant increases in frequency or severity of adverse effects seen between the exposed population and its appropriate control. Effects may be produced at this dose, but they are not considered to be adverse.

Octanol-Water Partition Coefficient (K_{OW})-The equilibrium ratio of the concentrations of a chemical in *n*-octanol and water, in dilute solution.

Odds Ratio-a means of measuring the association between an exposure (such as toxic substances and a disease or condition) which represents the best estimate of relative risk (risk as a ratio of the incidence among subjects exposed to a particular risk factor divided by the incidence among subjects who were not exposed to the risk factor). An odds ratio of greater than 1 is considered to indicate greater risk of disease in the exposed group compared to the unexposed.

Organophosphate or Orgauophosphorus Compound-a phosphorus containing organic compound and especially a pesticide that acts by inhibiting cholinesterase.

Permissible Exposure Limit (PEL)-An Occupational Safety and Health Administration (OSHA) allowable exposure level in workplace air averaged over an 8-hour shift of a 40 hour workweek.

Pesticide-general classification of chemicals specifically developed and produced for use in the control of agricultural and public health pests.

Pharmacokinetics-is the science of quantitatively predicting the fate (disposition) of an exogenous substance in an organism. Utilizing computational techniques, it provides the means of studying the absorption, distribution, metabolism and excretion of chemicals by the body.

Pharmacokinetic Model-is a set of equations that can be used to describe the time course of a parent chemical or metabolite in an animal system. There are two types of pharmacokinetic models: data-based and physiologically-based. A data-based model divides the animal system into a series of compartments which, in general, do not represent real, identifiable anatomic regions of the body whereby the physiologically-based model compartments represent real anatomic regions of the body.

Physiologically Based Pharmacodynamic (PBPD) Model-is a type of physiologically-based dose- response model which quantitatively describes the relationship between target tissue dose and toxic end points. These models advance the importance of physiologically based models in that they clearly describe the biological effect (response) produced by the system following exposure to an exogenous substance.

Physiologically Based Pharmacokinetic (PBPK) Model-is comprised of a series of compartments representing organs or tissue groups with realistic weights and blood flows. These models require a variety of physiological information: tissue volumes, blood flow rates to tissues, cardiac output, alveolar ventilation rates and, possibly membrane permeabilities. The models also use biochemical information such as air/blood partition coefficients, and metabolic parameters. PBPK models are also called biologically based tissue dosimetry models.

Prevalence-The number of cases of a disease or condition in a population at one point in time.

Prospective Study--a type of cohort study in which the pertinent observations are made on events occurring after the start of the study. A group is followed over time.

q_1*-The upper-bound estimate of the low-dose slope of the dose-response curve as determined by the multistage procedure. The q_1* can be used to calculate an estimate of carcinogenic potency, the incremental excess cancer risk per unit of exposure (usually μg/L. for water, mg/kg/day for food, and $\mu g/m^3$ for air).

Recommended Exposure Limit (REL)-A National Institute for Occupational Safety and Health (NIOSH) time-weighted average (TWA) concentrations for up to a l0-hour workday during a 40-hour workweek.

Reference Concentration (RfC)-An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious noncancer health effects during a lifetime. The inhalation reference concentration is for continuous inhalation exposures and is appropriately expressed in units of mg/m^3 or ppm.

Reference Dose (RFD)-An estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure of the human population to a potential hazard that is likely to be without risk of deleterious effects during a lifetime. The RfD is operationally derived from the No-Observed-Adverse-Effect Level (NOAEL- from animal and human studies) by a consistent application of uncertainty factors that reflect various types of data used to estimate RfDs and an additional modifying factor, which is based on a professional judgment of the entire database on the chemical. The RfDs are not applicable to nonthreshold effects such as cancer.

Reportable Quantity (RQ)-The quantity of a hazardous substance that is considered reportable under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA). Reportable quantities are (1) 1 pound or greater or (2) for selected substances, an amount established by regulation either under CERCLA or under Section 311 of the Clean Water Act. Quantities are measured over a 24-hour period.

Reproductive Toxicity-The occurrence of adverse effects on the reproductive system that may result from exposure to a chemical. The toxicity may be directed to the reproductive organs and/or the related endocrine system. The manifestation of such toxicity may be noted as alterations in sexual behavior, fertility, pregnancy outcomes, or modifications in other functions that are dependent on the integrity of this system.

Retrospective Study-A type of cohort study based on a group of people known to have been exposed at some time in the past. Data are collected from routinely recorded events, up to the time the study is undertaken. Retrospective studies are limited to casual factors that can be ascertained from existing records and/or examining survivors of the cohort.

Risk-the possibility or chance that some adverse effect will result from a given exposure to a chemical.

Risk Factor-An aspect of personal behavior or lifestyle, an environmental exposure, or an inborn or inherited characteristic, that is associated with an increased occurrence of disease or other health-related event or condition.

Risk Ratio-The ratio of the risk among persons with specific risk factors compared to the risk among persons without risk factors. A risk ratio greater than 1 indicates greater risk of disease in the exposed group compared to the unexposed.

Short-Term Exposure Limit (STEL)-The American Conference of Governmental Industrial Hygienists (ACGIH) maximum concentration to which workers can be exposed for up to 1.5 min continually. No more than four excursions are allowed per day, and there must be at least 60 min between exposure periods. The daily Threshold Limit Value - Time Weighted Average (TLV-TWA) may not be exceeded.

Target Organ Toxicity-This term covers a broad range of adverse effects on target organs or physiological systems (e.g., renal, cardiovascular) extending from those arising through a single limited exposure to those assumed over a lifetime of exposure to a chemical.

Teratogen-A chemical that causes structural defects that affect the development of an organism.

Threshold Limit Value (TLV)-An American Conference of Governmental Industrial Hygienists (ACGIH) concentration of a substance to which most workers can be exposed without adverse effect. The TLV may be expressed as a Time Weighted Average (TWA), as a Short-Term Exposure Limit (STEL), or as a ceiling limit (CL).

Time-Weighted Average (TWA)-An allowable exposure concentration averaged over a normal 8-hour workday or 40-hour workweek.

Toxic Dose$_{(50)}$ (TD$_{50}$)-A calculated dose of a chemical, introduced by a route other than inhalation, which is expected to cause a specific toxic effect in 50% of a defined experimental animal population.

Toxicokinetic-The study of the absorption, distribution and elimination of toxic compounds in the living organism.

Uncertainty Factor (UF)-A factor used in operationally deriving the Minimal Risk Level (MRL) or Reference Dose (RfD) or Reference Concentration (RfC) from experimental data. UFs are intended to account for (1) the variation in sensitivity among the members of the human population, (2) the uncertainty in extrapolating animal data to the case of human, (3) the uncertainty in extrapolating from data obtained in a study that is of less than lifetime exposure, and (4) the uncertainty in using Lowest-Observed-Adverse-Effect Level (LOAEL) data rather than No-Observed-Adverse-Effect Level (NOAEL) data. A default for each individual UF is 10; if complete certainty in data exists, a value of one can be used; however, a reduced UF of three may be used on a case-by-case basis, three being the approximate logarithmic average of 10 and 1.

Xenobiotic-any chemical that is foreign to the biological system.

APPENDIX A

MRLS AND CANCER CLASSIFICATION FOR TPH COMPONENTS AND WHOLE PRODUCTS

MRLs listed in Table A-l are found in Appendix A of the individual Toxicological Profile referenced. Appendix A of each profile describes the basis for ATSDR MRL's derivation and use and includes worksheets showing calculations used to derive each MRL.

APPENDIX A

Table A-1. Minimal Risk Levels and Cancer Classification for TPH Components and Whole Products[a]

Chemical	MRL (inhalation)	MRL (oral)	IARC Cancer Classification[b]	EPA Cancer classification[c]
Automotive gasoline[d]	NA	NA	NA	NA
Benzene[e]	0.05 ppm (acute), 0.004 ppm (intermediate)	NA	Group 1	Group A
Ethylbenzene[f]	0.2 ppm (intermediate)	NA	NA	Group D
Fuel Oils[g]	0.02 mg/m^3 (acute, diesel fuels), 0.01 mg/m^3 (intermediate, kerosene)	NA	Group 2A: petroleum refining, occupational Group 2B: marine diesel fuels & residual fuel oils Group 3: jet fuels & distillate diesel fuels	NA
n-Hexane[h]	0.6 ppm (chronic)	NA	NA	Group D
Jet Fuels[i]	9 mg/m^3 (intermediate, JP-4) 3 mg/m^3 (intermediate, JP 5/8) 0.3 mg/m^3 (chronic, JP-7)	NA	Group 3	NA
Mineral-based Crankcase Oil[j]	NA	NA	NA	NA
Naphthalene[k]	0.002 ppm (chronic, naphthalene)	0.05 mg/kg/day (acute, naphthalene) 0.02 mg/kg/day (intermediate, naphthalene) 0.07 mg/kg/day (chronic, 1-methylnaphthalene)	Group 3	Group D
PAHs[l]	NA	0.6 mg/kg/day (intermediate, acenaphthene) 0.4 mg/kg/day (intermediate, fluoranthene & fluorene) 10 mg/kg/day (intermediate, anthracene)	Group 2A: benz(a)anthracene, benzo(a)pyrene Group 2B: benzo(b)fluoranthene benzo(j)fluoranthene, ideno(1,2,3-c,d) pyrene Group 3: anthracene, benzo(g,h,i)perylene, & additional PAHs	Group B2: benz(a)anthracene, benzo(b)fluoranthene, benzo(a)pyrene, chrysene, dibenz(a,h)-anthracene, indeno(1,2,3-c,d)-pyrene Group D: acenaphthylene, anthracene, fluoranthene
Toluene[m]	3 ppm (acute) 1 ppm (chronic)	0.8 mg/kg/day (acute) 0.02 mg/kg/day (intermediate)	NA	Group D

APPENDIX A

Table A-1. Minimal Risk Levels and Cancer Classification for TPH Components and Whole Products[a] (continued)

Chemical	MRL (inhalation)	MRL (oral)	IARC Cancer Classification[b]	EPA Cancer classification[c]
Xylenes[n]	1 ppm (acute, mixed xylenes) 0.7 ppm (intermediate, mixed xylenes) 0.1 ppm (chronic, mixed xylenes)	1 mg/kg/day (acute, p-xylene) 0.2 mg/kg/day (intermediate, mixed xylenes) 0.6 mg/kg/day (intermediate, m-xylene)	Group 3	Group D

NA = not available

[a] MRLs listed in Table A-1 are found in Appendix A of the individual Toxicological Profile referenced. Appendix A of each profile describes the basis for ATSDR MRL's derivation and use and includes worksheets showing calculations used to derive each MRL.

[b] IARC Cancer Classifications: Group 1: carcinogenic to humans, Group 2A: probably carcinogenic to humans, Group 2B: possibly carcinogenic to humans, Group 3: not classifiable as to its carcinogenicity to humans

[c] EPA Cancer Classifications: Group A; known human carcinogen, Group B2: probable human carcinogen, Group C: possible human carcinogen, Group D; not classifiable as to its carcinogenicity to humans

[d] ATSDR. 1995a. Toxicological profile for automotive gasoline. Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA. NTIS PB95-264206.

[e] ATSDR. 1997a. Toxicological profile for benzene (update). Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

[f] ATSDR. 1997b. Toxicological profile for ethylbenzene (draft for public comment). Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

[g] ATSDR. 1995g. Toxicological profile for fuel oils. Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA. NTIS PB95-264222.

[h] ATSDR. 1997c. Toxicological profile for hexane (draft for public comment). Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

[i] ATSDR. 1995c. Toxicological profile for jet fuels (JP-4 and JP-7). Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA. NTIS PB95-264230.

ATSDR. 1998b. Toxicological profile for jet fuels (JP-5 and JP-8) (February 1998 draft final). Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

[j] ATSDR. 1997e. Toxicological profile for mineral-based crankcase oil. Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

[k] ATSDR. 1995e. Toxicological profile for naphthalene (update). Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

[l] ATSDR. 1995f. Toxicological profile for polycyclic aromatic hydrocarbons (PAHS) (update). Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

[m] ATSDR. 1994. Toxicological profile for toluene (update). Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA. TP-93/14.

[n] ATSDR. 1995d. Toxicological profile for xylenes (update). Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

APPENDIX B

USER'S GUIDE

Chapter 1

Public Health Statement

This chapter of the profile is a health effects summary written in non-technical language. Its intended audience is the general public especially people living in the vicinity of a hazardous waste site or chemical release. If the Public Health Statement were removed from the rest of the document, it would still communicate to the lay public essential information about the chemical.

The major headings in the Public Health Statement are useful to find specific topics of concern. The topics are written in a question and answer format. The answer to each question includes a sentence that will direct the reader to chapters in the profile that will provide more information on the given topic.

Chapter 6

Tables and Figures for Fraction-Specific Critical Effects

Tables (6-l through 6-l 1) and Figures (6-l through 6-16) summarize health effects and illustrate graphically levels of exposure associated with those effects. These levels cover health effects observed at increasing dose concentrations and durations, differences in response by species, minimal risk levels (MRLs) to humans for noncancer end points, and EPA's estimated range associated with an upper- bound individual lifetime cancer risk of 1 in 10,000 to 1 in 10,000,000. Use these tables and figures for a quick review of the health effects and to locate data for a specific exposure scenario. The Critical Effects tables and Exposure Assessment figures in Chapter 6 should always be used in conjunction with the text. All entries in these tables and figures represent studies that provide reliable, quantitative estimates of No-Observed-Adverse-Effect Levels (NOAELs), Lowest-Observed-Adverse-Effect Levels (LOAELs), or Cancer Effect Levels (CELs).

Chapter 6 (Section 6.7)

Relevance to Public Health

The Relevance to Public Health section provides a health effects summary based on evaluations of existing toxicologic, epidemiologic, and toxicokinetic information. This summary is designed to present interpretive, weight-of-evidence discussions for human health end points by addressing the following questions.

1 . What effects are known to occur in humans?

2 . What effects observed in animals are likely to be of concern to humans?

3 . What exposure conditions are likely to be of concern to humans, especially around hazardous waste sites?

The carcinogenic potential of the profiled substance is qualitatively evaluated, when appropriate, using existing toxicokinetic, genotoxic, and carcinogenic data. ATSDR does not currently assess cancer potency

or perform cancer risk assessments. Minimal risk levels (MRLs) for noncancer end points (if derived) and the end points from which they were derived are indicated and discussed.

Limitations to existing scientific literature that prevent a satisfactory evaluation of the relevance to public health are identified in the Data Needs section.

Interpretation of Minimal Risk Levels

Where sufficient toxicologic information is available, minimal risk levels (MRLs) for inhalation and oral routes of entry at each duration of exposure (acute, intermediate, and chronic) are provided. Though no new MRLs are derived for TPH, all available MRLs for TPH components and petroleum products are reviewed in Chapter 6 and presented in Appendix A. These MRLs are not meant to support regulatory action; but to acquaint health professionals with exposure levels at which adverse health effects are not expected to occur in humans. They should help physicians and public health officials determine the safety of a community living near a chemical emission, given the concentration of a contaminant in air or the estimated daily dose in water. MRLs are based largely on toxicological studies in animals and on reports of human occupational exposure.

MRL users should be familiar with the toxicologic information on which the number is based. In particular, the user should review the profile of the specific substance of concern (see Appendix A). Section 6.7, “Relevance to Public Health,” contains basic information known about the substance. Other sections such as 6.9, “Interactions with Other Substances,” and 6.10, “Populations that are Unusually Susceptible” provide important supplemental information.

MRL users should also understand the MRL derivation methodology. MRLs are derived using a modified version of the risk assessment methodology the Environmental Protection Agency (EPA) provides (Barnes and Dourson 1988) to determine reference doses for lifetime exposure (RfDs).

To derive an MRL, ATSDR generally selects the most sensitive end point which, in its best judgement, represents the most sensitive human health effect for a given exposure route and duration. ATSDR cannot make this judgement or derive an MRL unless information (quantitative or qualitative) is available for all potential systemic, neurological, and developmental effects. If this information and reliable quantitative data on the chosen end point are available, ATSDR derives an MRL using the most sensitive species (when information from multiple species is available) with the highest NOAEL that does not exceed any adverse effect levels. When a NOAEL is not available, a lowest-observed-adverse-effect level (LOAEL) can be used to derive an MRL, and an uncertainty factor (UP) of 10 must be employed. Additional uncertainty factors of 10 must be used both for human variability to protect sensitive subpopulations (people who are most susceptible to the health effects caused by the substance) and for interspecies variability (extrapolation from animals to humans). In deriving an MRL, these individual uncertainty factors are multiplied together. The product is then divided into the inhalation concentration or oral dosage selected from the study. Uncertainty factors used in developing a substance-specific MRL are provided in the footnotes of the LSE Tables in the profiles listed in Appendix A.

The section covers end points in the same order they appear within the Discussion of Health Effects by Route of Exposure section, by route (inhalation, oral, dermal) and within route by effect. Human data are presented first, then animal data. Both are organized by duration (acute, intermediate, chronic). *In vitro* data and data from parenteral routes (intramuscular, intravenous, subcutaneous, etc.) are also considered in this section. If data are located in the scientific literature, a table of genotoxicity information is included.

APPENDIX C

ACRONYMS, ABBREVIATIONS, AND SYMBOLS

ACGIH	American Conference of Governmental Industrial Hygienists
ADME	Absorption, Distribution, Metabolism, and Excretion
atm	atmosphere
ASTM	American Society for Testing and Materials
ATSDR	Agency for Toxic Substances and Disease Registry
BCF	bioconcentration factor
BSC	Board of Scientific Counselors
BTEX	benzene, toluene, ethylbenzene, xylene
C	Centigrade
CDC	Centers for Disease Control and Prevention
CEL	Cancer Effect Level
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
CLP	Contract Laboratory Program
cm	centimeter
CNS	central nervous system
d	day
DHEW	Department of Health, Education, and Welfare
DHHS	Department of Health and Human Services
DNAPL	denser nonaqueous phase liquids
DOL	Department of Labor
EC	equivalent carbon number
ECG	electrocardiogram
EEG	electroencephalogram
EPA	Environmental Protection Agency
EKG	see ECG
DRO	diesel range organics
F	Fahrenheit
F_1	first filial generation
FAO	Food and Agricultural Organization of the United Nations
FEMA	Federal Emergency Management Agency
FID	flame ionization detection
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
fpm	feet per minute
ft	foot
FR	*Federal Register*
g	gram
GC	gas chromatography
GC/MS	gas chromatography/mass spectrometry
gen	generation
GRO	gasolines range organics
HEC	human equivalent concentration
HPLC	high-performance liquid chromatography
hr	hour
HSSM	hydrocarbon spill screening model
IDLH	Immediately Dangerous to Life and Health
IARC	International Agency for Research on Cancer

IOC	index of concern
ILO	International Labor Organization
in	inch
Kd	adsorption ratio
kg	kilogram
kkg	metric ton
K_{oc}	organic carbon partition coefficient
K_{ow}	octanol-water partition coefficient
L	liter
LC	liquid chromatography
LC_{Lo}	lethal concentration, low
LC_{50}	lethal concentration, 50% kill
LD_{Lo}	lethal dose, low
LD_{50}	lethal dose, 50% kill
LNAPL	lighter nonaqueous phase liquids
LOAEL	lowest-observed-adverse-effect level
LSE	Levels of Significant Exposure
LUST	leaking underground storage tanks
m	meter
MADEP	Massachusetts Department of Environmental Protection
MARPOL	International Convention for the Prevention of Pollution Ships (marine pollution)
mg	milligram
min	minute
mL	milliliter
mm	millimeter
mmHg	millimeters of mercury
MTBE	methyl-*tert*-butyl ether
mmol	millimole
mo	month
mppcf	millions of particles per cubic foot
MRL	Minimal Risk Level
MS	mass spectrometry
NAPL	non-aqueous phase liquids
NIEHS	National Institute of Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NIOSHTIC	NIOSH's Computerized Information Retrieval System
ng	nanogram
nm	nanometer
NHANES	National Health and Nutrition Examination Survey
nmol	nanomole
NOAEL	no-observed-adverse-effect level
NOES	National Occupational Exposure Survey
NOHS	National Occupational Hazard Survey
NPDES	National Pollutant Discharge Elimination System
NPL	National Priorities List
NRC	National Research Council
NTIS	National Technical Information Service
NTP	National Toxicology Program
OSHA	Occupational Safety and Health Administration
PAHs	polyaromatic hydrocarbons
PCBs	polychlorinated biphenyls

PEL	permissible exposure limit
pg	picogram
PID	photo ionization detector
pmol	picomole
PHS	Public Health Service
PMR	proportionate mortality ratio
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion
RBCA	Risk-Based Corrective Action
RBSL	risk-based screening level
RCRA	Resource Conservation and Recovery Act
REL	recommended exposure limit
RfC	Reference Concentration
RfD	Reference Dose
RP	relative potency
RQ	reportable quantity
RTECS	Registry of Toxic Effects of Chemical Substances
sec	second
SCE	sister chromatid exchange
SFE	supercritical fluid extraction
SIC	Standard Industrial Classification
SMR	standard mortality ratio
spcc	spill prevention, control and countermeasure
SPE	solid phase extraction
SSTL	site-specific target level
STEL	short term exposure limit
STORET	STORAGE and RETRIEVAL
TOG	total recoverable oil and grease
TPH	total petroleum hydrocarbons
TPHCWG	Total Petroleum Hydrocarbons Criteria Working Group
TRPH	total recoverable petroleum hydrocarbons
TSCA	Toxic Substances Control Act (TSCA)
TLV	threshold limit value
TSCA	Toxic Substances Control Act
TSDF	treatment, storage and disposal facilities, hazardous wastes
TRI	Toxics Release Inventory
TWA	time-weighted average
U.S.	United States
UF	uncertainty factor
UST	underground storage tank
yr	year
WHO	World Health Organization
wk	week
WOE	weight-of-evidence, classification of carcinogenicity

APPENDIX C

>	greater than
≥	greater than or equal to
=	equal to
<	less than
≤	less than or equal to
%	percent
α	alpha
β	beta
δ	delta
γ	gamma
μm	micrometer
μg	microgram

APPENDIX D

PETROLEUM PRODUCT COMPOSITION

Petroleum products can be made up of hundreds of individual petroleum hydrocarbons. TPH is a value that represents the amount of petroleum hydrocarbons in a given sample, as previously presented in Section 2.1. There are far more than 250 individual chemicals that are known as petroleum hydrocarbons and there is an inherent complexity involved in chemically and physically describing and categorizing these components.

This Appendix contains a list of the more prominent individual chemicals that are likely to be associated with TPH. Table D-1 sorts the petroleum hydrocarbons into groups based upon basic chemical structure. An "ATSDR Fraction" column identifies which one of six particular fractions individual chemicals have been assigned to for health effects purposes, as described in Section 6.1.3 and shown below.

Aliph1 = Aliphatics EC_5-EC_8

Aliph2 = Aliphatics $EC_{>8}$-EC_{16}

Aliph3 = Aliphatics $EC_{>16}$-EC_{35}

Arom1 = Aromatics EC_5-EC_9

Arom2 = Aromatics $EC_{>9}$-EC_{16}

Arom3 = Aromatics $EC_{>16}$-EC_{35}

APPENDIX D

Table D-1. Petroleum Product Composition

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
Straight Chain Alkanes						
Propane	3	3		0.01–0.14	Gasoline	LUFT 1988
n-Butane	4	4		3.93–4.70	Gasoline	LUFT 1988
				0.12	JP-4	API 1993
n-Pentane	5	5	*Aliph1*	5.75–10.92	Gasoline	LUFT 1988
				1.06	JP-4	API 1993
n-Hexane	6	6	*Aliph1*	0.24–3.50	Gasoline	LUFT 1988
				0.7–1.8	Crude Oil	API 1993
				2.21	JP-4	API 1993
n-Heptane	7	7	*Aliph1*	0.31–1.96	Gasoline	LUFT 1988
				0.8–2.3	Crude Oil	API 1993
				3.67	JP-4	API 1993
				0.03	JP-8	API 1993
				0.1	Kerosene	API 1993
n-Octane	8	8	*Aliph1*	0.36–1.43	Gasoline	LUFT 1988
				0.9–1.9	Crude Oil	API 1993
				3.8	JP-4	API 1993
				0.12	JP-5	API 1993
				0.9	JP-8	API 1993
				0.2–0.3	Kerosene	API 1993
				0.1	Diesel	BP 1996
				0.1	Fuel Oil #2	BP 1996

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
n-Nonane	9	9	*Aliph2*	0.07–0.83	Gasoline	LUFT 1988
				0.6–1.9	Crude Oil	API 1993
				2.25	JP-4	API 1993
				0.38	JP-5	API 1993
				0.31	JP-8	API 1993
				0.4–0.8	Kerosene	API 1993
				0.19–0.49	Diesel	BP 1996
				0.20–0.30	Fuel Oil #2	BP 1996
n-Decane	10	10	*Aliph2*	0.04–0.50	Gasoline	LUFT 1988
				1.8	Crude Oil	API 1993
				2.16	JP-4	API 1993
				1.79	JP-5	API 1993
				1.31	JP-8	API 1993
				1.5–1.7	Kerosene	API 1993
				0.28–1.2	Diesel	BP 1996
				0.5	Fuel Oil #2	BP 1996
n-Undecane	11	11	*Aliph2*	0.05–0.22	Gasoline	LUFT 1988
				1.7	Crude Oil	API 1993
				2.32	JP-4	API 1993
				3.95	JP-5	API 1993
				4.13	JP-8	API 1993
				3.5–6.1	Kerosene	API 1993
				0.57–2.3	Diesel	BP 1996
				0.80–0.90	Fuel Oil #2	BP 1996

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
n-Dodecane	12	12	*Aliph2*	0.04–0.09	Gasoline	LUFT 1988
				1.7	Crude Oil	API 1993
				2	JP-4	API 1993
				3.94	JP-5	API 1993
				4.72	JP-8	API 1993
				2.8–5.7	Kerosene	API 1993
				1.0–2.5	Diesel	BP 1996
				0.84–1.20	Fuel Oil #2	BP 1996
n-Tridecane	13	13	*Aliph2*	1.52	JP-4	API 1993
				3.45	JP-5	API 1993
				4.43	JP-8	API 1993
				3.1–5.2	Kerosene	API 1993
				1.5–2.8	Diesel	BP 1996
				0.96–2.00	Fuel Oil #2	BP 1996
n-Tetradecane	14	14	*Aliph2*	0.73	JP-4	API 1993
				2.72	JP-5	API 1993
				2.99	JP-8	API 1993
				2.3–4.7	Kerosene	API 1993
				0.61–2.7	Diesel	BP 1996
				1.03–2.50	Fuel Oil #2	BP 1996
n-Pentadecane	15	15	*Aliph2*	1.67	JP-5	API 1993
				1.61	JP-8	API 1993
				0.6–2.3	Kerosene	API 1993
				1.9–3.1	Diesel	BP 1996
				1.13–3.20	Fuel Oil #2	BP 1996

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Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
n-Hexadecane	16	16	*Aliph2*	1.07	JP-5	API 1993
				0.45	JP-8	API 1993
				0.1–0.7	Kerosene	API 1993
				1.5–2.8	Diesel	BP 1996
				1.05–3.30	Fuel Oil #2	BP 1996
n-Heptadecane	17	17	*Aliph3*	0.12	JP-5	API 1993
				0.08	JP-8	API 1993
				0.4	Kerosene	API 1993
				1.4–2.9	Diesel	BP 1996
				0.65–3.60	Fuel Oil #2	BP 1996
n-Octadecane	18	18	*Aliph3*	0.02	JP-8	API 1993
				0.3	Kerosene	API 1993
				1.2–2.0	Diesel	BP 1996
				0.55–2.50	Fuel Oil #2	BP 1996
n-Nonadecane	19	19	*Aliph3*	0.2	Kerosene	API 1993
				0.7–1.5	Diesel	BP 1996
				0.33–1.30	Fuel Oil #2	BP 1996
n-Eicosane	20	20	*Aliph3*	0.1	Kerosene	API 1993
				0.4–1.0	Diesel	BP 1996
				0.18–0.60	Fuel Oil #2	BP 1996
n-Heneicosane	21	21	*Aliph3*	0.1	Kerosene	API 1993
				0.26–0.83	Diesel	BP 1996
				0.09–0.40	Fuel Oil #2	BP 1996
n-Docosane	22	22	*Aliph3*	0.14–0.44	Diesel	BP 1996

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
				0.1	Fuel Oil #2	BP 1996
n-Tetracosane	24	24	*Aliph3*	0.35	Diesel	BP 1996
n-Hexacosane	26	26	*Aliph3*			
Branched Chain Alkanes						
Isobutane	4	3.67		0.12–0.37	Gasoline	LUFT 1988
				0.66	JP-4	API 1993
2,2-Dimethylbutane	6	5.37	*Aliph1*	0.17–0.84	Gasoline	LUFT 1988
				0.04	Crude Oil	API 1993
				0.1	JP-4	API 1993
2,3-Dimethylbutane	6	5.68	*Aliph1*	0.59–1.55	Gasoline	LUFT 1988
				0.04–0.14	Crude Oil	API 1993
2,2,3-Trimethyl-butane	7	6.36	*Aliph1*	0.01–0.04	Gasoline	LUFT 1988
2,2,3,3-Tetra-methylbutane	8	7.3	*Aliph1*	0.24	JP-4	API 1993
Neopentane	5	4.32		0.02–0.05	Gasoline	LUFT 1988
Isopentane	5	4.75		6.07–10.17	Gasoline	LUFT 1988
2-Methylpentane	6	5.72	*Aliph1*	2.91–3.85	Gasoline	LUFT 1988
				0.3–0.4	Crude Oil	API 1993
				1.28	JP-4	API 1993
3-Methylpentane	6	5.85	*Aliph1*	2.4 (vol)	Gasoline	LUFT 1988
				0.3–0.4	Crude Oil	API 1993
				0.89	JP-4	API 1993
3-Ethylpentane	7		*Aliph1*	0.05	Crude Oil	API 1993
2,2-Dimethylpentane	7	6.25	*Aliph1*	0.25	JP-4	API 1993
2,4-Dimethylpentane	7	6.31	*Aliph1*	0.23–1.71	Gasoline	LUFT 1988
				0.05	Crude Oil	API 1993

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
2,3-Dimethylpentane	7	6.69	*Aliph1*	0.32–4.17	Gasoline	LUFT 1988
				0.1–0.6	Crude Oil	API 1993
3,3-Dimethylpentane	7	6.55	*Aliph1*	0.02–0.03	Gasoline	LUFT 1988
2,2,3-Trimethyl-pentane	8	7.37	*Aliph1*	0.09–0.23	Gasoline	LUFT 1988
2,2,4-Trimethyl-pentane	8	6.89	*Aliph1*	0.32–4.58	Gasoline	LUFT 1988
				0.004	Crude Oil	API 1993
2,3,3-Trimethyl-pentane	8	7.58	*Aliph1*	0.05–2.28	Gasoline	LUFT 1988
				0.006	Crude Oil	API 1993
2,3,4-Trimethyl-pentane	8	7.55	*Aliph1*	0.11–2.80	Gasoline	LUFT 1988
				0.005	Crude Oil	API 1993
2-Methyl-3-ethyl-pentane	8	7.66	*Aliph1*	0.04	Crude Oil	API 1993
2,4-Dimethyl-3-ethylpentane	9		*Aliph2*	0.03–0.07	Gasoline	LUFT 1988
2-Methylhexane	7	6	*Aliph1*	68 0.36–1.48	Gasoline	LUFT 1988
				0.7	Crude Oil	API 1993
				2.35	JP-4	API 1993
3-Methylhexane	7	6.76	*Aliph1*	0.30–1.77	Gasoline	LUFT 1988
				0.19–0.5	Crude Oil	API 1993
				1.97	JP-4	API 1993
2,2-Dimethylhexane	8	7.25	*Aliph1*	0.01–0.1	Crude Oil	API 1993
				0.71	JP-4	API 1993
2,3-Dimethylhexane	8	7.65	*Aliph1*	0.06–0.16	Crude Oil	API 1993
2,4-Dimethylhexane	8	7.38	*Aliph1*	0.34–0.82	Gasoline	LUFT 1988
				0.06	Crude Oil	API 1993
				0.58	JP-4	API 1993
2,5-Dimethylhexane	8	7.36	*Aliph1*	0.24–0.52	Gasoline	LUFT 1988

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
				0.06	Crude Oil	API 1993
				0.37	JP-4	API 1993
3,3-Dimethylhexane	8	7.45	*Aliph1*	0.03	Crude Oil	API 1993
				0.26	JP-4	API 1993
3,4-Dimethylhexane	8	7.74	*Aliph1*	0.16–0.37	Gasoline	LUFT 1988
3-Ethylhexane	8	7.79	*Aliph1*	0.01	Gasoline	LUFT 1988
2-Methyl-3-ethyl-hexane	9			0.04–0.13	Gasoline	LUFT 1988
2,2,4-Trimethyl-hexane	9	7.93	*Aliph1*	0.11–0.18	Gasoline	LUFT 1988
2,2,5-Trimethyl-hexane	9	7.87	*Aliph1*	0.17–5.89	Gasoline	LUFT 1988
2,3,3-Trimethyl-hexane	9			0.05–0.12	Gasoline	LUFT 1988
2,3,5-Trimethyl-hexane	9	8.24	*Aliph2*	0.05–1.09	Gasoline	LUFT 1988
2,4,4-Trimethyl-hexane	9	8.07		0.02–0.16	Gasoline	LUFT 1988
2-Methylheptane	8	7.71	*Aliph1*	0.48–1.05	Gasoline	LUFT 1988
				2.7	JP-4	API 1993
3-Methylheptane	8	7.78	*Aliph1*	0.63–1.54	Gasoline	LUFT 1988
				3.04	JP-4	API 1993
4-Methylheptane	8	7.72	*Aliph1*	0.22–0.52	Gasoline	LUFT 1988
				0.92	JP-4	API 1993
2,2-Dimethylheptane	9	8.28	*Aliph2*	0.01–0.08	Gasoline	LUFT 1988
2,3-Dimethylheptane	9	8.64	*Aliph2*	0.13–0.51	Gasoline	LUFT 1988
				0.05	Crude Oil	API 1993
2,4-Dimethylheptane	9	8.34	*Aliph2*	0.43	JP-4	API 1993
2,5-Dimethylheptane	9	8.47	*Aliph2*	0.52	JP-4	API 1993

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
2,6-Dimethylheptane	9	8.47	*Aliph2*	0.07–0.23	Gasoline	LUFT 1988
				0.05 -0.25	Crude Oil	API 1993
3,3-Dimethylheptane	9	8.42		0.01–0.08	Gasoline	LUFT 1988
3,4-Dimethylheptane	9	8.62	*Aliph2*	0.07–0.33	Gasoline	LUFT 1988
2,2,4-Trimethyl-heptane	10		*Aliph2*	0.12–1.70	Gasoline	LUFT 1988
2,4,6-Trimethyl-heptane	10		*Aliph2*	0.07	JP-5	API 1993
3,3,5-Trimethyl-heptane	10		*Aliph2*	0.02–0.06	Gasoline	LUFT 1988
3-Ethylheptane	9	8.77	*Aliph2*	0.02–0.16	Gasoline	LUFT 1988
4-Ethylheptane	9	8.69	*Aliph2*	0.18	JP-4	API 1993
2-Methyloctane	9		*Aliph2*	0.14–0.62	Gasoline	LUFT 1988
				0.4	Crude Oil	API 1993
				0.88	JP-4	API 1993
3-Methyloctane	9	8.78	*Aliph2*	0.34–0.85	Gasoline	LUFT 1988
				0.1–0.4	Crude Oil	API 1993
				0.79	JP-4	API 1993
				0.07	JP-5	API 1993
				0.04	JP-8	API 1993
4-Methyloctane	9	8.71	*Aliph2*	0.11–0.55	Gasoline	LUFT 1988
				0.1	Crude Oil	API 1993
				0.86	JP-4	API 1993
2,6-Dimethyloctane	10	9.32	*Aliph2*	0.06–0.12	Gasoline	LUFT 1988
2-Methylnonane	10	9.72	*Aliph2*	0.06–0.41	Gasoline	LUFT 1988
3-Methylnonane	10	9.78	*Aliph2*	0.06–0.32	Gasoline	LUFT 1988
4-Methylnonane	10		*Aliph2*	0.04–0.26	Gasoline	LUFT 1988
4-Methyldecane	11		*Aliph2*	0.78	JP-5	API 1993
2-Methyldecane	11		*Aliph2*	0.61	JP-5	API 1993

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
				0.41	JP-8	API 1993
2,6-Dimethyldecane	12		*Aliph2*	0.72	JP-5	API 1993
				0.66	JP-8	API 1993
2-Methylundecane	12		*Aliph2*	0.64	JP-4	API 1993
				1.39	JP-5	API 1993
				1.16	JP-8	API 1993
3-Methylundecane	12		*Aliph2*	0.09–0.28	Diesel	BP 1996
2-Methyldodecane	13		*Aliph2*	0.15–0.52	Diesel	BP 1996
2,6-Dimethyl-undecane	13		*Aliph2*	0.71	JP-4	API 1993
				2	JP-5	API 1993
				2.06	JP-8	API 1993
3-Methyltridecane	14		*Aliph2*	0.13–0.30	Diesel	BP 1996
2-Methyltetradecane	15		*Aliph2*	0.34–0.63	Diesel	BP 1996
Cycloalkanes						
Cyclopentane	5	5.66	*Aliph1*	0.19–0.58	Gasoline	LUFT 1988
				0.05	Crude Oil	API 1993
Methylcyclopentane	6	6.27	*Aliph1*	not quantified	Gasoline	LUFT 1988
				0.3–0.9	Crude Oil	API 1993
				1.16	JP-4	API 1993
1-Methyl-cis-2-ethyl-cyclopentane	8		*Aliph1*	0.06–0.11	Gasoline	LUFT 1988
1-Methyl-trans-3-ethylcyclopentane	8		*Aliph1*	0.06–0.12	Gasoline	LUFT 1988
1,1-Dimethylcyclo-pentane	7	6.72	*Aliph1*	0.06–0.2	Crude Oil	API 1993
1-cis-2-Dimethyl-cyclo-pentane	7	7.21	*Aliph1*	0.07–0.13	Gasoline	LUFT 1988
				0.54	JP-4	API 1993
1-Trans-2-dimethyl-cyclo-pentane	7	6.87	*Aliph1*	0.06–0.20	Gasoline	LUFT 1988

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
				0.15–.5	Crude Oil	API 1993
1-cis-3-Dimethyl-cyclopentane	7	6.82	*Aliph1*	0.2	Crude Oil	API 1993
				0.34	JP-4	API 1993
1-Trans-3-dimethyl-cyclopentane	7	6.85	*Aliph1*	0.2–0.9	Crude Oil	API 1993
				0.36J	P-4	API 1993
1,1,2-Trimethyl-cyclopentane	8	7.67	*Aliph1*	0.06–0.1	Gasoline	LUFT 1988
				0.06	Crude Oil	API 1993
1,1,3-Trimethyl-cyclopentane	8	7.25	*Aliph1*	0.3	Crude Oil	API 1993
1-Trans-2-cis-3-tri-methylcyclopentane	8	7.51	*Aliph1*	0.01–0.25	Gasoline	LUFT 1988
				0.3–0.4	Crude Oil	API 1993
1-Trans-2-cis-4-tri-methylcyclo-pentane	8			0.03–0.16	Gasoline	LUFT 1988
				0.2	Crude Oil	API 1993
1-Trans-2-trans-4-tri-methylcyclopentane	8	7.19	*Aliph1*			
Ethylcyclopentane	7	7.34	*Aliph1*	0.14–0.21	Gasoline	LUFT 1988
				0.26	JP-4	API 1993
n-Propylcyclo-pentane	8	7.1	*Aliph1*	0.01–0.06	Gasoline	LUFT 1988
Isopropylcyclo-pentane	8		*Aliph1*	0.01–0.02	Gasoline	LUFT 1988
1-cis-3-Dimethyl-cyclohexane	8	7.75	*Aliph1*	0.42	JP-4	API 1993
1-Trans-2-dimethyl-cyclohexane	8	7.94	*Aliph1*	0.3	Crude Oil	API 1993
1-Trans-3-dimethyl-cyclohexane	8	7.99	*Aliph1*	0.05–0.12	Gasoline	LUFT 1988
1,4-Dimethylcyclo-hexane	8					

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Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
Ethylcyclohexane	8	8.38	*Aliph2*	0.17–0.42	Gasoline	LUFT 1988
				0.2	Crude Oil	API 1993
Cyclohexane	6	6.59	*Aliph1*	0.7	Crude Oil	API 1993
				0.08	Gasoline	API 1993
				1.24	JP-4	API 1993
Methylcyclohexane	7	7.22	*Aliph1*	2.27	JP-4	API 1993
1-Methyl-2-ethyl-cyclohexane	9		*Aliph2*	0.39	JP-4	API 1993
1-Methyl-3-ethyl-cyclohexane	9		*Aliph2*	0.17	JP-4	API 1993
1-Methyl-4-ethyl-cyclohexane	9		*Aliph2*	0.48	JP-5	API 1993
				0.1	JP-8	API 1993
1,3,5-Trimethyl-cyclohexane	9		*Aliph2*	0.99	JP-4	API 1993
				0.09	JP-5	API 1993
				0.06	JP-8	API 1993
1,1,3-Trimethyl-cyclo-hexane	9	8.45	*Aliph2*	0.48	JP-4	API 1993
				0.05	JP-5	API 1993
				0.06	JP-8	API 1993
n-Butylcyclohexane	10		*Aliph2*	0.7	JP-4	API 1993
				0.9	JP-5	API 1993
				0.74	JP-8	API 1993
n-Propylcyclohexane	9		*Aliph2*	0.14	JP-8	API 1993
Hexylcyclohexane	12		*Aliph2*	0.93	JP-8	API 1993
Heptylcyclohexane	13		*Aliph2*	0.99	JP-5	API 1993
				1	JP-8	API 1993
Pentylcyclopentane	10	10.4	*Aliph2*			
1-Trans-2-trans-4-tri-methylcyclo-hexane	9		*Aliph2*	0.2	Crude Oil	API 1993

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
Straight Chained Alkenes						
Propylene	3					
cis-2-Butene	4	4.25		0.13–0.17	Gasoline	LUFT 1988
trans-2-Butene	4	4.1		0.16–0.20	Gasoline	LUFT 1988
Pentene-1	5	4.89		0.33–0.45	Gasoline	LUFT 1988
1-Pentyne	5	5.13	*Aliph1*			
cis-2-Pentene	5	5.16	*Aliph1*	0.43–0.67	Gasoline	LUFT 1988
Trans-2-pentene	5	5.08	*Aliph1*	0.52–0.90	Gasoline	LUFT 1988
1-Hexene	6	5.9	*Aliph1*			
1-Hexyne	6	6.09	*Aliph1*			
cis-2-Hexene	6	6.14	*Aliph1*	0.15–0.24	Gasoline	LUFT 1988
Trans-2-hexene	6	6.05	*Aliph1*	0.18–0.36	Gasoline	LUFT 1988
cis-3-Hexene	6	6.03	*Aliph1*	0.11–0.13	Gasoline	LUFT 1988
Trans-3-hexene	6	6.02	*Aliph1*	0.12–0.15	Gasoline	LUFT 1988
cis-3-Heptene	7	7.01	*Aliph1*	0.14–0.17	Gasoline	LUFT 1988
Trans-2-heptene	7	7.05	*Aliph1*	0.06–0.10	Gasoline	LUFT 1988
1-Octene	8	7.89	*Aliph1*			
1-Nonene	9	8.69	*Aliph2*			
1-Decene	10	9.91	*Aliph2*			
Tridecene	13		*Aliph2*	0.45	JP-5	API 1993
				0.73	JP-8	API 1993
Branched Chain Alkenes						
2-Methyl-1-butene	5	4.96		0.22–0.66	Gasoline	LUFT 1988
3-Methyl-1-butene	5	4.57		0.08–0.12	Gasoline	LUFT 1988
2-Methyl-2-butene	5	5.21	*Aliph1*	0.96–1.28	Gasoline	LUFT 1988
2,3-Dimethyl-1-butene	6	5.7	*Aliph1*	0.08–0.10	Gasoline	LUFT 1988
2-Methyl-1-pentene	6	5.89	*Aliph1*	0.20–0.22	Gasoline	LUFT 1988

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
2,3-Dimethyl-1-pentene	7		*Aliph1*	0.01–0.02	Gasoline	LUFT 1988
2,4-Dimethyl-1-pentene	7	6.48	*Aliph1*	0.02–0.03	Gasoline	LUFT 1988
4,4-Dimethyl-1-pentene	7		*Aliph1*	0.60 (vol)	Gasoline	LUFT 1988
2-Methyl-2-pentene	6	6.07	*Aliph1*	0.27–0.32	Gasoline	LUFT 1988
3-Methyl-cis-2-pentene	6	6.11	*Aliph1*	0.35–0.45	Gasoline	LUFT 1988
3-Methyl-trans-2-pentene	6	6.22	*Aliph1*	0.32–0.44	Gasoline	LUFT 1988
4-Methyl-cis-2-pentene	6	5.69	*Aliph1*	0.04–0.05	Gasoline	LUFT 1988
4-Methyl-trans-2-pentene	6	5.73	*Aliph1*	0.08–0.30	Gasoline	LUFT 1988
4,4-Dimethyl-cis-2-pentene	7	6.47	*Aliph1*	0.02	Gasoline	LUFT 1988
4,4-Dimethyl-trans-2-pentene	7	6.23	*Aliph1*	Not quantified	Gasoline	LUFT 1988
3-Ethyl-2-pentene	7	7.07	*Aliph1*	0.03–0.04	Gasoline	LUFT 1988
Cycloalkenes						
Cyclopentene	5	5.55	*Aliph1*	0.12–0.18	Gasoline	LUFT 1988
3-Methylcyclo-pentene	6	6.1	*Aliph1*	0.03–0.08	Gasoline	LUFT 1988
Cyclohexene	6	6.74	*Aliph1*	0.03	Gasoline	LUFT 1988
Alkyl Benzenes						
Benzene	6	6.5	*Arom1*	0.12–3.50	Gasoline	LUFT 1988
				0.04–0.4	Crude Oil	API 1993
				0.5	JP-4	API 1993
				0.003–0.10	Diesel	BP 1996
				<0.125	Fuel Oil #2	BP 1996

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
Toluene	7	7.58	*Arom1*	2.73–21.80	Gasoline	LUFT 1988
				0.09–2.5	Crude Oil	API 1993
				1.33	JP-4	API 1993
				0.007–0.70	Diesel	BP 1996
				0.025–0.110	Fuel Oil #2	BP 1996
Ethylbenzene	8	8.5	*Arom1*	0.36–2.86	Gasoline	LUFT 1988
				0.09–0.31	Crude Oil	API 1993
				0.37	JP-4	API 1993
				0.007–0.20	Diesel	BP 1996
				0.028–0.04	Fuel Oil #2	BP 1996
o-Xylene	8	8.81	*Arom1*	0.68–2.86	Gasoline	LUFT 1988
				0.03–0.68	Crude Oil	API 1993
				1.01	JP-4	API 1993
				0.09	JP-5	API 1993
				0.06	JP-8	API 1993
				0.001–0.085	Diesel	BP 1996
m-Xylene	8	8.6	*Arom1*	1.77–3.87	Gasoline	LUFT 1988
				0.08–2.0	Crude Oil	API 1993
				0.96	JP-4	API 1993
				0.13	JP-5	API 1993
				0.06	JP-8	API 1993
				0.018–0.512	Diesel	BP 1996
p-Xylene	8	8.61	*Arom1*	0.77–1.58	Gasoline	LUFT 1988
				0.09–0.68	Crude Oil	API 1993
				0.35	JP-4	API 1993
				0.018–0.512	Diesel	BP 1996
Styrene	9	8.83	*Arom1*	<.002	Diesel	BP 1996

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
1-Methyl-4-ethyl-benzene	9	9.57	*Arom2*	0.18–1.00	Gasoline	LUFT 1988
				0.03–0.13	Crude Oil	API 1993
				0.43	JP-4	API 1993
1-Methyl-2-ethyl-benzene	9	9.71	*Arom2*	0.19–0.56	Gasoline	LUFT 1988
				0.01–0.09	Crude Oil	API 1993
				0.23	JP-4	API 1993
1-Methyl-3-ethyl-benzene	9	9.55	*Arom2*	0.31–2.86	Gasoline	LUFT 1988
				0.04–0.4	Crude Oil	API 1993
				0.49	JP-4	API 1993
1-Methyl-2-n-propyl-benzene	10		*Arom2*	0.01–0.17	Gasoline	LUFT 1988
1-Methyl-3-n-propyl-benzene	10		*Arom2*	0.08–0.56	Gasoline	LUFT 1988
1-Methyl-2-isopropylbenzene	10		*Arom2*	0.01–0.12	Gasoline	LUFT 1988
				0.29	JP-4	API 1993
				0.56	JP-8	API 1993
1-Methyl-3-isopropylbenzene	10		*Arom2*	10.09		
1-Methyl-4-isopropylbenzene	10	10.13	*Arom2*	0.003–0.026	Diesel	BP 1996
1-Methyl-3-t-butyl-benzene	11		*Arom2*	0.03–0.11	Gasoline	LUFT 1988
1-Methyl-4-t-butyl-benzene	11	10.92	*Arom2*	0.04–0.13	Gasoline	LUFT 1988
1,2-Dimethyl-3-ethylbenzene	10	10.93	*Arom2*	0.02–0.19	Gasoline	LUFT 1988
1,2-Dimethyl-4-ethylbenzene	10	10.75	*Arom2*	0.50–0.73	Gasoline	LUFT 1988
				0.77	JP-4	API 1993

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
1,3-Dimethyl-2-ethyl-benzene	10	10.81	*Arom2*	0.21–0.59	Gasoline	LUFT 1988
1,3-Dimethyl-4-ethylbenzene	10	10.75	*Arom2*	0.03–0.44	Gasoline	LUFT 1988
1,3-Dimethyl-5-ethylbenzene	10	10.51	*Arom2*	0.11–0.42	Gasoline	LUFT 1988
				0.61	JP-4	API 1993
				0.62	JP-8	API 1993
1,3-Dimethyl-5-t-butylbenzene	12		*Arom2*	0.02–0.16	Gasoline	LUFT 1988
1,4-Dimethyl-2-ethylbenzene	10	10.68	*Arom2*	0.05–0.36	Gasoline	LUFT 1988
				0.7	JP-4	API 1993
1,2,3-Trimethyl-benzene	9	10.06	*Arom2*	0.21–0.48	Gasoline	LUFT 1988
				0.1	Crude Oil	API 1993
1,2,4-Trimethyl-benzene	9	9.84	*Arom2*	0.66–3.30	Gasoline	LUFT 1988
				0.13–0.69	Crude Oil	API 1993
				1.01	JP-4	API 1993
				0.37	JP-5	API 1993
				0.27	JP-8	API 1993
1,3,5-Trimethyl-benzene	9	9.62	*Arom2*	0.13–1.15	Gasoline	LUFT 1988
				0.05–0.18	Crude Oil	API 1993
				0.42	JP-4	API 1993
				0.09–0.24	Diesel	BP 1996
1,2,3,4-Tetramethyl-benzene	10	11.57	*Arom2*	0.02–0.19	Gasoline	LUFT 1988
				0.2	Crude Oil	API 1993
1,2,3,5-Tetramethyl-benzene	10	11.09	*Arom2*	0.14–1.06	Gasoline	LUFT 1988

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
1,2,4,5-Tetramethyl-benzene	10	11.05	*Arom2*	0.05–0.67	Gasoline	LUFT 1988
1,2-Diethylbenzene	10	10.52	*Arom2*	0.57	Gasoline	LUFT 1988
1,3-Diethylbenzene	10	10.4	*Arom2*	0.05–0.38	Gasoline	LUFT 1988
				0.46	JP-4	API 1993
				0.61	JP-5	API 1993
1,4-Diethylbenzene	10	10.46	*Arom2*	0.77	JP-5	API 1993
1,2,4-Triethyl-benzene	12	12.29	*Arom2*	0.72	JP-5	API 1993
				0.99	JP-8	API 1993
1,3,5-Triethyl-benzene	12	12.1	*Arom2*	0.6	JP-8	API 1993
n-Propylbenzene	9	9.47	*Arom2*	0.08–0.72	Gasoline	LUFT 1988
				0.71	JP-4	API 1993
				0.03–0.048	Diesel	BP 1996
Isopropylbenzene	9	9.13	*Arom2*	<10.01–0.23	Gasoline	LUFT 1988
				0.3	JP-4	API 1993
				<0.01	Diesel	BP 1996
n-Butylbenzene	10	10.5	*Arom2*	0.04–0.44	Gasoline	LUFT 1988
				0.031–0.046	Diesel	BP 1996
Isobutylbenzene	10	9.96	*Arom2*	0.01–0.08	Gasoline	LUFT 1988
sec-Butylbenzene	10	9.98	*Arom2*	0.01–0.13	Gasoline	LUFT 1988
t-Butylbenzene	10	9.84	*Arom2*	0.12	Gasoline	LUFT 1988
1-t-Butyl-3,4,5-trimethyl-benzene	13		*Arom2*	0.24	JP-5	API 1993
n-Pentylbenzene	11	11.49	*Arom2*	0.01–0.14	Gasoline	LUFT 1988
Isopentylbenzene	11		*Arom2*	0.07–0.17	Gasoline	LUFT 1988
n-Hexylbenzene	12	12.5	*Arom2*			
n-Heptylbenzene	13		*Arom2*	0.27	JP-5	API 1993

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
				0.25	JP-8	API 1993
n-Octylbenzene	14		*Arom2*	0.78	JP-5	API 1993
				0.61	JP-8	API 1993
Biphenyl	12	14.26	*Arom2*	0.006–.04	Crude Oil	API 1993
				0.7	JP-5	API 1993
				0.63	JP-8	API 1993
				0.01–0.12	Diesel	BP 1996
				0.006–0.009	Fuel Oil #2	BP 1996
4-Methylbiphenyl	13	14.92	*Arom2*			
4,4'-Dimethyl-biphenyl	14	16.55	*Arom3*			
Phenylcyclohexane	12		*Arom2*	0.82	JP-5	API 1993
				0.87	JP-8	API 1993
Naphtheno-Benzenes						
Acenaphthene	12	15.5	*Arom2*	0.013–0.022	Fuel Oil #2	BP 1996
Acenaphthylene	12	15.06	*Arom2*	0.006	Fuel Oil #2	BP 1996
Indan	9	10.27	*Arom2*	0.25–0.34	Gasoline	LUFT 1988
				0.07	Crude Oil	API 1993
1-Methylindan	10		*Arom2*	0.04–0.17	Gasoline	LUFT 1988
2-Methylindan	10	11.39	*Arom2*	0.02–0.10	Gasoline	LUFT 1988
4-Methylindan	10	11.33	*Arom2*	0.01–0.16	Gasoline	LUFT 1988
5-Methylindan	10	11.28	*Arom2*	0.09–0.30	Gasoline	LUFT 1988
Tetralin (tetrahydro-naphthalene)	10	11.7	*Arom2*	0.01–0.14	Gasoline	LUFT 1988
				0.03	Crude Oil	API 1993
5-Methyl-thtrohydro-naphthalene	11		*Arom2*	0.08	Crude Oil	API 1993

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
6-Methylthtrohydr-naphthalene	11		*Arom2*	0.09	Crude Oil	API 1993
Fluorene	13	16.55	*Arom3*	0.003–0.06	Crude Oil	API 1993
				0.034–0.15	Diesel	BP 1996
				0.004–0.045	Fuel Oil #2	BP 1996
1-Methylfluorene	14	17.99	*Arom3*			
Fluoranthene	16	21.85	*Arom3*	0.0000007–0.02	Diesel	BP 1996
				0.000047–0.00037	Fuel Oil #2	BP 1996
2,3-Benzofluorene	17	23.83	*Arom3*			
1,2-Benzofluorene	17	24.2	*Arom3*	<0.0024	Fuel Oil #2	BP 1996
Benzo(a)fluorene	17		*Arom3*	<0.0006	Fuel Oil #2	BP 1996
Benzo(ghi)fluor-anthene	18		*Arom3*	<0.0024	Fuel Oil #2	BP 1996
Benz(b)fluoranthene	20	30.14	*Arom3*	0.0000003–0.000194	Diesel	BP 1996
				<0.0024	Fuel Oil #2	BP 1996
Benz(k)fluoranthene	20	30.14	*Arom3*	0.0000003–0.000195	Diesel	BP 1996
				<0.00006	Fuel Oil #2	BP 1996
Indeno (1,2,3-cd) pyrene	22	35.01	*Arom3*	0.000001–0.000097	Diesel	BP 1996
				<0.0012	Fuel Oil #2	BP 1996
Alkyl Naphthalenes						
Naphthalene	10	11.69	*Arom2*	0.09–0.49	Gasoline	LUFT 1988
				0.02–0.09	Crude Oil	API 1993
				0.5	JP-4	API 1993
				0.57	JP-5	API 1993

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
				1.14	JP-8	API 1993
				0.01–0.80	Diesel	BP 1996
				0.009–0.40	Fuel Oil #2	BP 1996
1-Methyl-naphthalene	11	12.99	*Arom2*	0.78	JP-4	API 1993
				1.44	JP-5	API 1993
				1.84	JP-8	API 1993
				0.001–0.81	Diesel	BP 1996
				0.29–0.48	Fuel Oil #2	BP 1996
2-Methyl-naphthalene	11	12.84	*Arom2*	0.56	JP-4	API 1993
				1.38	JP-5	API 1993
				1.46	JP-8	API 1993
				0.001–1.49	Diesel	BP 1996
				0.36 -1.00	Fuel Oil #2	BP 1996
1,3-Dimethyl-naphthalene	12	14.77	*Arom2*	0.55–1.28	Diesel	BP 1996
1,4-Dimethyl-naphthalene	12	14.6	*Arom2*	0.110–0.23	Diesel	BP 1996
				0.043–0.045	Fuel Oil #2	BP 1996
1,5-Dimethyl-naphthalene	12	13.87	*Arom2*	0.16–0.36	Diesel	BP 1996
2,3-Dimethyl-naphthalene	12	15	*Arom2*	0.46	JP-5	API 1993
				0.36	JP-8	API 1993
2,6-Dimethyl-naphthalene	12	14.6	*Arom2*	0.25	JP-4	API 1993
				1.12	JP-5	API 1993
				1.34	JP-8	API 1993

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
1-Ethylnaphthalene	12	14.41	*Arom2*	0.32	JP-5	API 1993
				0.33	JP-8	API 1993
2-Ethylnaphthalene	12	13.99	*Arom2*			
1,4,5-Trimethyl-naphthalene	13	10.6	*Arom2*			
1-Phenyl-naphthalene	16		*Arom3*			
Polynuclear Aromatics						
Anthracene	14	19.43	*Arom3*	0.000003 -0.02	Diesel	BP 1996
				0.00010–0.011	Fuel Oil #2	BP 1996
2-Methyl anthracene	15	20.73	*Arom3*	0.000015–0.018	Diesel	BP 1996
				0.009–0.017	Fuel Oil #2	BP 1996
9-Methyl anthracene	15	20.45	*Arom3*			
2-Ethyl anthracene	16		*Arom3*			
9,10-Dimethyl anthracene	16		*Arom3*	0.002–0.006	Fuel Oil #2	BP 1996
Phenanthrene	14	19.36	*Arom3*	0.003–0.05	Crude Oil	API 1993
				0.000027–0.30	Diesel	BP 1996
				0.009 -0.170	Fuel Oil #2	BP 1996
1-Methyl-phenanthrene	15	20.73	*Arom3*	0.000011–0.024	Diesel	BP 1996
				0.017	Fuel Oil #2	BP 1996
2-Methyl-phenanthrene	15		*Arom3*	0.014–0.18	Diesel	BP 1996
				0.768	Fuel Oil #2	BP 1996
3-Methyl-phenanthrene	15		*Arom3*	0.000013–0.011	Diesel	BP 1996
4- & 9-Methyl-phenanthrene	15		*Arom3*	0.00001–0.034	Diesel	BP 1996

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
Pyrene	16	20.8	*Arom3*	Not quantified	Gasoline	LUFT 1988
				0.000018–0.015	Diesel	BP 1996
				0.00–0.012	Fuel Oil #2	BP 1996
1-Methylpyrene	17		*Arom3*	0.0000024–0.00137	Diesel	BP 1996
2-Methylpyrene	17		*Arom3*	0.0000037–0.00106	Diesel	BP 1996
Benz(a)anthracene	18	26.37	*Arom3*	Not quantified	Gasoline	LUFT 1988
				0.0000021–0.00067	Diesel	BP 1996
				0.000002–0.00012	Fuel Oil #2	BP 1996
Chrysene	18	27.41	*Arom3*	0.000045	Diesel	BP 1996
				0.000037–0.00039	Fuel Oil #2	BP 1996
Triphenylene	18	26.61	*Arom3*	0.00033	Diesel	BP 1996
				0.00002–0.00014	Fuel Oil #2	BP 1996
Cyclopenta(cd)-pyrene	18		*Arom3*	0.000002–0.0000365	Diesel	BP 1996
1-Methyl-7-isopropyl-phenanthrene	18		*Arom3*	0.0000015–0.00399	Diesel	BP 1996
3-Methylchrysene	19		*Arom3*	<0.001	Diesel	BP 1996
5-Methylchrysene	19		*Arom3*			
6-Methylchrysene	19		*Arom3*	<0.0005	Diesel	BP 1996
Benzo(a)pyrene	20	31.34	*Arom3*	0.000019–0.00028	Gasoline	LUFT 1988
				0.000005–0.00084	Diesel	BP 1996
				0.000001–0.000060	Fuel Oil #2	BP 1996
Benz(e)pyrene	20	31.17	*Arom3*	Not quantified	Gasoline	LUFT 1988

APPENDIX D

Table D-1. Petroleum Product Composition (continued)

Compound	Carbon Number	EC[a]	ATSDR Fraction[b]	Weight Percent	Fuel Type	Reference
				0.0000054–0.000240	Diesel	BP 1996
				0.0000020–0.000010	Fuel Oil #2	BP 1996
Benzo(ghi)pyrene	20		*Arom3*	0.0000010–0.0000070	Fuel Oil #2	BP 1996
Perylene	20	31.34	*Arom3*	<0.0001	Diesel	BP 1996
				<0.0024	Fuel Oil #2	BP 1996
3-Methyl-cholanthrene	21		*Arom3*	<0.00006	Fuel Oil #2	BP 1996
Benzo(b)chrysene	22		*Arom3*	<0.0036	Fuel Oil #2	BP 1996
Benz(ghi)perylene	22	34.01	*Arom3*	Not quantified	Gasoline	LUFT 1988
				0.0000009–0.00004	Diesel	BP 1996
				20.0000057	Fuel Oil #2	BP 1996
Picene	22		*Arom3*	0.0000004–0.000083	Diesel	BP 1996
				<0.00012	Fuel Oil #2	BP 1996
1,2,5,6-Dibenz-anthracene	22	33.92	*Arom3*			
Coronene	24	34.01	*Arom3*	<0.000024	Fuel Oil #2	BP 1996

[a] Effective Carbon Number Index

[b] Aliph1 = Aliphatics EC_5-EC_8; Arom1 = Aromatics EC_5-EC_9
Aliph2 = Aliphatics $EC_{>8}$-EC_{16}; Arom2 = Aromatics $EC_{>9}$-EC_{16}
Aliph3 = Aliphatics $EC_{>16}$-EC_{35}; Arom3 = Aromatics $EC_{>16}$-EC_{35}

Source: Total Petroleum Hydrocarbons Criteria Working Group. 1997. Selection of Representative TPH Fractions Based on Fate and Transport Considerations, vol. 3.

APPENDIX E

IDENTITY, COMPONENTS, AND CHEMICAL/PHYSICAL PROPERTIES OF SELECTED PETROLEUM PRODUCTS

TPH is a value that represents the amount of petroleum hydrocarbons in a given sample, as previously presented in Section 2.1. There are far more than 250 individual chemicals that are known as petroleum hydrocarbons. Some of these are listed in Appendix D. More often, many of the petroleum hydrocarbons are known by the names associated with the more common whole petroleum products, such as gasoline, fuel oil, mineral oil, and jet fuels, for example. These whole products are actually mixtures of numerous individual compounds, such as those listed in Appendix D, as well as, sometimes, non-petroleum hydrocarbon additives. Because of the complexity involved in chemically and physically describing and categorizing these whole products this Appendix contains more detailed information about the more prominent products that are likely to be associated with TPH. A list of the information is given below.

E-1. Automotive Gasoline
- A. Chemical Identity
- B. Composition
- C. Chemical and Physical Properties

E-2. Stoddard Solvent
- A. Chemical Identity
- B. Composition
- C. Chemical and Physical Properties

E-3. JP-4
- A. Chemical Identity
- B. Composition
- C. Chemical and Physical Properties

E-4. Fuel Oil
- A. Chemical Identity
- B. Composition
- C. Chemical and Physical Properties

E-5. Crankcase Oils, Mineral-based
- A. Chemical Identity
- B. Composition
- C. Chemical and Physical Properties

E-6. Mineral Oil
- A. Chemical Identity
- B. Chemical and Physical Properties

APPENDIX E

Table E-1.a. Chemical Identity of Gasoline[a]

Character	Information	Reference
Chemical Name	Gasoline	RTECS 1995
Synonym(s)	Casing head gasoline, natural gasoline petrol, motor fuel, motor spirit	RTECS 1995; ATSDR 1995a
Identification Numbers:		
CAS Registry	8006-61-9	RTECS 1995
NIOSH RTECS	LX3300000	RTECS 1995
EPA Hazardous Waste	No data	
OHM/TADS	7217073	OHM/TADS 1991
DOT/UN/NA/IMCO shipping	UN1203, UN1257	RTECS 1995
HSDB	No data	
NCI	No data	

[a] Gasoline is a mixture of C-4 through C-12 hydrocarbons, primarily consisting of 4–8% alkanes, 2–5% alkenes, 25–40% isoalkanes, 3–7% cycloalkanes, 1–4% cycloalkenes, and 20–50% aromatics.

Source: ATSDR (1995a)

CAS = Chemical Abstracts Services; DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; EPA = Environmental Protection Agency; HSDB = Hazardous Substances Data Bank; NCI = National Cancer Institute; NIOSH = National Institute for Occupational Safety and Health; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data Systems; RTECS = Registry of Toxic Effects of Chemical Substances

APPENDIX E

Table E-1.b. Major Hydrocarbon Components of Gasoline

Fraction Compound	Gasoline Weight Percent Range	Gasoline Weight Percent Mean
C6 Aromatics		
Benzene	0.12–3.50	2.34
C3–C6 Aliphatic		
Propane	0.01–0.14	0.00666
n-Butane	3.93–4.70	3.57
Isobutane	0.12–0.37	0.316
n-Pentane	5.75–10.92	3.18
n-Hexane	0.24–3.50	2.61
2,2-Dimethylbutane	0.17–0.84	0.304
2,3-Dimethylbutane	0.59–1.55	1.41
Neopentane	0.02-0.05	
Isopentane	6.07–10.17	6.22
2-Methylpentane	2.91–3.85	3.35
3-Methylpentane	2.4 (vol)	2.14
Cyclopentane	0.19–0.58	0.131
Methylcyclopentane	not quantified	2.08
Cyclohexane		0.722
1-Pentene	0.33–0.45	0.222
1-Pentyne		
cis-2-Pentene	0.43–0.67	0.865
1-Hexene		0.22
1-Hexyne		
3-Methyl-1-butene	0.08–0.12	0.417
2-Methyl-1-Pentene	0.20–0.22	0.258
Cyclopentene	0.12–0.18	0.236
Cyclohexene	0.03	

APPENDIX E

Table E-1.b. Major Hydrocarbon Components of Gasoline (continued)

Fraction Compound	Gasoline Weight Percent Range	Gasoline Weight Percent Mean
C7–C8 Aromatics		
Toulene	2.73–21.80	8.21
Ethylbenzene	0.36–2.86	1.9
o-Xylene	0.68–2.86	2.71
m-Xylene	1.77–3.87	3.5
p-Xylene	0.77–1.58	3.5
Styrene		
C7–C8 Aliphatics		
n- Heptane	0.31–1.96	1.14
n-Octane	0.36–1.43	0.426
2,2,3-Trimethylbutane	0.01–0.04	0.025
2,2-Dimethylpentane	0.25	0.0878
2,4-Dimethylpentane	0.23–1.71	0.734
2,3-Dimethylpentane	0.32–4.17	1.54
3,3-Dimethylpentane	0.02–0.03	0.0989
2,2,4-Trimethylpentane	0.32–4.58	1.64
2,3,4-Trimethylpentane	0.11–2.80	0.519
2-Methylhexane	0.36–1.48	1.44
3-Methylhexane	0.30–1.77	1.5
2-Methylheptane	0.48–1.05	0.614
3-Methylheptane	0.63–1.54	0.647
1,1,3-Trimethycyclo-pentane	0.3	0.0511
1-Trans-2-trans-4-Trimethylcyclopentane		
n-Propylcyclopentane	0.01–0.06	
1-Trans-2-dimethyl-cyclohexane		
1-Trans-4-dimethylcyclohexane		0.142
Methylcyclohexane		0.611

Table E-1.b. Major Hydrocarbon Components of Gasoline (continued)

Fraction Compound	Gasoline Weight Percent Range	Gasoline Weight Percent Mean
Trans-2-heptene	0.06–0.10	0.105
1-Octene		0.101
C9–C10 Aromatics		
1-Methyl-4-ethylbenzene	0.18–1.00	0.837
1-Methyl-2-ethylbenzene	0.19–0.56	2.89
1-Methyl-4-isopropylbenzene		
1,2,3-trimethylbenzene	0.21–0.48	0.766
1,2,4-trimethylbenzene	0.66–3.30	3.41
1,3,5-trimethylbenzene	0.13–1.15	1.14
1,2,4,5-tetramethyl-benzene	0.05–0.67	
n-Propylbenzene	0.08–0.72	0.648
Isopropylbenzene	<0.01–0.23	
n-Butylbenzene	0.04–0.44	
Isobutylbenzene	0.01–0.08	
sec-Butylbenzene	0.01–0.13	
t-Butylbenzene	0.12	
Indan	0.25–0.34	
Tetralin (tetrahydronaphthalene)	0.01–0.14	
Naphthalene	0.09–0.49	
C9–C10 Aliphatics		
n-Nonane	0.07–0.83	0.243
n-Decane	0.04–0.50	0.26
2,2,5-Trimethylhexane	0.17–5.89	0.177
4-Methyloctane	0.11–0.55	0.5
1,1,3-Trimethylcyclo-hexane		
Pentylcyclopentane		
1-Nonene		
1-Decene		

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Table E-1.b. Major Hydrocarbon Components of Gasoline (continued)

Fraction Compound	Gasoline Weight Percent Range	Gasoline Weight Percent Mean
C11–C12 Aromatics		
n-Pentylbenzene	0.01–0.14	
n-Hexylbenzene		
Biphenyl		
Acenaphthene		
Acenaphthylene		
1-Methylnaphthalene		
1,4-Dimethyl-naphthalene		
2,3-Dimethyl-naphthalene		
2,6-Dimethylnaphthalene		
1-Ethylnaphthalene		
2-Ethylnaphthalene		
C11-C12 Aliphatics		
n-Undecane	0.05–0.22	
n-Dodecane	0.04–0.09	
C13-C16 Aromatics		
Fluorene		
Fluoranthene		
1,4,5-Trimethylnaphthalene		
Anthracene		
9-Methyl anthracene		
Phenanthrene		
Pyrene	Not quantified	
C13–C16 Aliphatics		
n-Tetradecane		
n-Hexadecane		
C17–and up Aromatics		
Benz(k)fluoranthene		

Table E-1.b. Major Hydrocarbon Components of Gasoline (continued)

Fraction Compound	Gasoline Weight Percent Range	Gasoline Weight Percent Mean
Benz(a)anthracene	Not quantified	
Chrysene		
Triphenylene		
Benzo(a)pyrene	0.19–2.8 mg/kg	
Benz(e)pyrene	Not quantified	
Perylene		
3-Methylcholanthrene		
Benz(ghi)perylene	Not quantified	
1,2,5,6-dibenz anthracene		
C17–and up Aliphatics		
n-Octadecane		
n-Eicosane		

Source: Table taken from EA Engineering 1995, *Total Petroleum Hydrocarbon Criteria, Working Group Project #3, Based on Fate and Transport Considerations,* Prepared for Armstrong Laboratory, Brooks Air Force Base, Occupational Medicine, Brooks Air Force Base, Texas, Prepared by EA Engineering, Science, and Technology, Lafayette, California.

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Table E-1c. Physical and Chemical Properties of Gasoline

Property	Information	Reference
Molecular weight	108[a]	ATSDR 1995a
Color	Colorless to pale brown	Sax and Lewis 1989
Physical state	Liquid	Sax and Lewis 1989
Melting point	No data	
Boiling point	Initially, 39 °C After 10% distilled, 60 °C After 50% distilled, 110 °C After 90% distilled, 170 °C Final boiling point, 204 °C	Sax and Lewis 1989
Density	0.7–0.8 g/cm^3 [b]	IARC 1998
Odor	Gasoline	Weiss 1986
Odor threshold	0.025 ppm [c]	Weiss 1986
Solubility		
Water at 20 °C	Insoluble	Sax and Lewis 1989
Organic solvent(s)	Absolute alcohol, ether, chloroform, benzene	Sax and Lewis 1989
Partition coefficients		
Log K_{ow}	2.13–4.87[d]	U.S. Air Force 1989
Log K_{oc}	1.81–4.56[d]	U.S. Air Force 1989
Vapor pressure[e]		
at 60 °C	465 mm Hg	ASTM 1989
at 56 °C	518 mm Hg	
at 51 °C	593 mm Hg	
at 47 °C	698 mm Hg	
at 41 °C	773 mm Hg	
Henry's law constant at 20 °C	4.8x10^{-4} m^3/mol[d]	U.S. Air Force 1989
Autoignition temperature	280–486 °C	Sax and Lewis 1989; Weiss
Flashpoint	-46 °C	Sax and Lewis 1989
Flammability limits	1.4–7.4%	Weiss 1986
Conversion factors	No data	
Explosive limits	1.3–6.0%	Sax and Lewis 1989

[a] Average molecular weight
[b] Temperature not specified
[c] Not specified whether data for air or water
[d] Since data are not available for gasoline, ranges are given indicating different values for the individual components.
[e] The American Society for Testing and Materials (ASTM) has established guidelines on compositions of gasoline that will permit satisfactory performance under varying conditions. These guidelines define 5 volatility classes that vary by seasonal climatic changes. The values given for vapor pressure at the given temperatures are based on these volatility classes.

Table E-2.a. Chemical Identity of Stoddard Solvent[a]

Character	Information	Reference
Chemical Name	Stoddard solvent	ATSDR 1995b
Synonym(s)	Dry cleaning safety solvent, naphtha safety solvent, PD-680, petroleum solvent, spotting naphtha, varnoline, white spirits	ATSDR 1995b; U.S. Air Force 1989
Registered Trade Name(s)	Texsolve S, Varsol 1	ATSDR 1995b
Identification Numbers:		
CAS Registry	8052-41-3	ATSDR 1995b
NIOSH RTECS	WJ8925000	ATSDR 1995b
EPA Hazardous Waste	No data	
OHM/TADS	No data	
DOT/UN/NA/IMCO shipping	1268 27	ATSDR 1995b
HSDB	No data	
NCI	No data	

[a] Stoddard solvent is a mixture of C-7 through C-12 hydrocarbons primarily containing 30-50% linear and branched alkanes, 30–40% cycloalkanes, and 10–20% aromatic hydrocarbons.

CAS = Chemical Abstracts Services; DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; EPA = Environmental Protection Agency; HSDB = Hazardous Substances Data Bank; NCI = National Cancer Institute
NIOSH = National Institute for Occupational Safety and Health; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data Systems; RTECS = Registry of Toxic Effects of Chemical Substances

Table E-2.b. Possible Formulations of Stoddard Solvent (Percent)

Hydrocarbons	Stoddard solvent[a] (regular)	Stoddard solvent[a] (140 flash)	Stoddard solvent[b]	Stoddard solvent[c]	Stoddard solvent[d]
Alkanes (paraffins)	30–50 (48 average)	60.8	34.9	41.6	47.7
n-Nonane					
n-Decane					
Methylnonanes					
2,6-Dimethyloctane					
n-Undecane					
Dodecanes					
Terdecanes					
Others					
Cycloalkanes (cycloparaffins)	30–40 (38 average)	35.7		39.5	37.6
Monocycloparaffins		24.5	34.9	27.9	26
Trimethylcyclohexane					
tert-Butylcyclohexane					
n-Butycyclopentane					
n-Butycyclohexane					
Other Cycloparaffins					
Dicycloparaffins		11.2	5	11.6	11.6
Tricycloparaffins			0.4	0.00	
Acenaphthenes			0.4		

Table E-2.b. Possible Formulations of Stoddard Solvent (Percent) (continued)

Hydrocarbons	Stoddard solvent[a] (regular)	Stoddard solvent[a] (140 flash)	Stoddard solvent[b]	Stoddard solvent[c]	Stoddard solvent[d]
Aromatics	10–20 (14.1 average)	3.4		18.9	
Alkylbenzenes	14	3.03	22	17.6	14.1
Dimethylethylbenzenes					
n-Propylbenzene					
Ethyltoluenes					
1,2,4-Trimethylbenzene					
Other aromatics			1.1		
Other benzenes	0.1	0.07			0.1
Indans/Tetralins	<1	0.3	1.8	1.3	0.5
Indenes			0.1		
Naphthalenes			0.2		
Acenaphthalenes			0.3		
Tricyclicaromatics			0.1		

[a] Adapted from Air Force (1989)
[b] Adapted from American Petroleum Institute (1976)
[c] Adapted from Suntech Group (1978); API 1978
[d] Adapted from Carpenter et al. (1975); this paper also includes a mass spectral analysis of components by carbon number within a hydrocarbon class, e.g.; C_8 alkanes

Table E-2.c. Physical and Chemical Properties of Stoddard Solvent

Property	Information	Reference
Molecular weight	144 (mean); 135–145 (range)	Air Force 1989b; Carpenter et al. 1975b
Color	Clear, colorless	Sax and Lewis 1989
Physical state	Liquid	Sax and Lewis 1989
Melting point	No data	
Boiling point	154–202 °C 160–199 °C	Air Force 1989b Coast Guard 1985
Density at 20 °C	0.78 g/mL	NIOSH 1990
Odor	Similar to kerosene	NIOSH 1990
Odor threshold	0.9 ppm (5.1 mg/m^3) 2 mg/m^3	Carpenter et al. 1975b Hastings et al. 1984
Solubility: Water Organic solvents	 Insoluble Absolute alcohol, benzene, ether, chloroform, carbon tetrachloride, carbon disulfide	 McDermott 1975 Sax and Lewis 1989
Partition coefficients: Log K_{ow} Log K_{oc}	 3.16–7.06 2.85–6.74	Air Force 1989b Air Force 1989b
Vapor pressure at 25 °C	4–4.5 mm Hg	McDermott 1975
Henry's law constant at 20 °C	4.4x10^0 atm-m^3/mol	Air Force 1989b
Autoignition temperature	232 °C	Sax and Lewis 1989
Flashpoint	37.8–60.0 °C 38–43 °C	Air Force 1989b Sax and Lewis 1989
Flammability limits in air at 25 °C	0.9–6.0	Carpenter et al. 1975b
Conversion factors: at 25 °C and 760 mm	 1 mg/L = 174.5 ppm 1 ppm = 5.73 mg/m^3	 Carpenter et al. 1975b Air Force 1989b
Explosive limits Lower limit Upper limit	 0.9% 6%	McDermott 1975

Table E-3a. Chemical Identity, Composition and Chemical Physical Properties of JP-4[a]

Character	Information	Reference
Chemical Name	JP-4	OHM/TADS 1985
Synonym(s)	Jet Fuel-4	OHM/TADS 1985
Registered Trade Name(s)	NIL-T-5624-L-Amd. 1; wide cut; JP-4 military (gasoline type)	Air Force 1990c; Dickson and Woodard 1987; Dukek 1978; IARC 1989
Identification Numbers:		
CAS Registry	50815-00-4	OHM/TADS 1985
NIOSH RTECS	NY9340000	RTECS 1991
EPA Hazardous Waste	No data	
OHM/TADS	7217071	OHM/TADS 1985
DOT/UN/NA/IMCO shipping	1863	CHRIS 1986
HSDB	No data	
NCI	No data	

[a]JP-4 is a mixture of C-4 to C-16 hydrocarbons with an approximate distribution by chemical class of 32% straight alkanes, 31% branched alkanes, 16% cycloalkanes, and 21% aromatic hydrocarbons.

CAS = Chemical Abstracts Services; DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; EPA = Environmental Protection Agency; HSDB = Hazardous Substances Data Bank; NCI = National Cancer Institute; NIOSH = National Institute for Occupational Safety and Health; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data Systems; RTECS = Registry of Toxic Effects of Chemical Substances

Table E-3.b. Typical Hydrocarbon Composition of JP-4 Jet Fuel

Compound	Weight Percent
Straight Alkanes	32
Butane	0.12
Pentane	1.06
Hexane	2.21
Heptane	3.67
Octane	3.80
Nonane	2.25
Decane	2.16
Undecane	2.32
Dodecane	2.00
Tridecane	1.52
Tetradecane	0.73
Pentadecane	—
Hexadecane	—
Heptadecane	—
Octadecane	—
Isoalkanes	31
Isobutane	0.66
2,2-Dimethylbutane	0.10
2-Methylpentane	1.28
3-Methylpentane	0.89
2,2-Dimethylpentane	0.25
2-Methylhexane	2.35
3-Methylhexane	1.97
2,2,3,3-Tetramethylbutane	0.24
2,5-Dimethylhexane	0.37
2,4-Dimethylhexane	0.58
3,3-Dimethylhexane	0.26
2,2-Dimethylhexane	0.71

Table E-3.b. Typical Hydrocarbon Composition of JP-4 Jet Fuel (continued)

Compound	Weight Percent
2-Methylheptane	2.70
4-Methylheptane	0.92
3-Methylheptane	3.04
2,5-Dimethylheptane	0.52
2,4-Dimethylheptane	0.43
4-Ethylheptane	0.18
4-Methyloctane	0.86
2-Methyloctane	0.88
3-Methyloctane	0.79
2-Methylundecane	0.64
2,6-Dimethylundecane	0.71
2,4,6-Trimethylheptane	—
4-Methyldecane	—
2-Methyldecane	—
2,6-Dimethyldecane	—
2-Methylundecane	—
2,6-Dimethylundecane	—
Cycloalkanes	16
Methylcyclopentane	1.16
Cyclohexane	1.24
trans-1,3-Dimethylcyclopentane	0.36
cis-1,3-Dimethylcyclopentane	0.34
cis-1,2-Dimethylcyclopentane	0.54
Methylcyclohexane	2.27
Ethylclopentane	0.26
1,2,4-Trimethylcyclopentane	0.25
1,2,3-Trimethylcyclopentane	0.25
cis-1,3-Dimethylcyclohexane	0.42

Table E-3.b. Typical Hydrocarbon Composition of JP-4 Jet Fuel (continued)

Compound	Weight Percent
1-Methyl-3-ethylcyclohexane	0.17
1-Methyl-2-ethylcyclohexane	0.39
Dimethylcyclohexane	0.43
1,3,5-Trimethylcyclohexane	0.99
1,1,3-Trimethylcyclohexane	0.48
1-Methyl-4-ethylcyclohexane	0.48
n-Butylcyclohexane	0.70
Propylcyclohexane	—
Hexylcyclohexane	—
Heptylcyclohexane	—
Aromatic Hydrocarbons	21
Benzene	0.50
Toluene	1.33
Ethylbenzene	0.37
m-Xylene	0.96
p-Xylene	0.35
o-Xylene	1.01
Isopropylbenzene	0.30
n-Propylbenzene	0.71
1-Methyl-3-ethylbenzene	0.49
1-Methyl-4-ethylbenzene	0.43
1,3,5-Trimethylbenzene	0.42
1-Methyl-2-ethylbenzene	0.23
1,2,4-Trimethylbenzene	1.01
1,3-Diethylbenzene	0.46
1,4-Diethylbenzene	—
1-Methyl-4-propylbenzene	0.40
1,3-Dimethyl-5-ethylbenzene	0.61

Table E-3.b. Typical Hydrocarbon Composition of JP-4 Jet Fuel (continued)

Compound	Weight Percent
1-Methyl-2-isopropylbenzene	0.29
1,4-Dimethyl-2-ethylbenzene	0.70
1,2-Dimethyl-4-ethylbenzene	0.77
1,2,3,4-Tetramethylbenzene	0.75
1-Ethylpropylbenzene	—
1,2,4-Triethylbenzene	—
1,3,5-Triethylbenzene	—
Phenylcyclohexane	—
1-tert-Butyl-3,4,5-trimethylbenzene	—
n-Heptylbenzene	—
Naphthalene	0.50
2-Methylnaphthalene	0.56
1-Methylnaphthalene	0.78
2,6-Dimethylnaphthalene	0.25
Biphenyl	—
1-Ethylnaphthalene	—
2,3-Dimethylnaphthalene	—
n-Octylbenzene	—

Table E-3.c. Physical and Chemical Properties of JP-4[a]

Property	Information	Reference
Molecular weight	Not applicable[b]	
Color	Colorless to straw colored	CHRIS 1986; Martel 1992
Physical state	Liquid	CHRIS 1986
Melting point	-46 °C -40–72 °C	OHM/TADS 1985 ITC 1985
Boiling point (1 atm)	50–270 °C 90–300 °C 45–280 °C	Air Force 1989b ITC 1985 Dickson and Woodward 1987
Density at 15 °C	751–802 kg/m^3 (specification)	
Odor	Like gasoline and/or kerosene	
Odor threshold:		
Water	No data	
Air	1 ppm	CHRIS 1986
Solubility:		
Water at 20 °C	57 mg/L	CRC 1984
Organic solvents	Since many of the components are organic solvents, the fuel is generally miscible with organic solvents	ITC 1985
Partition coefficients:		
Log K_{ow}	Major components range from 3 to 4.5	ITC 1985
Log K_{oc}	No data	
Vapor pressure at 20 °C	91 mm Hg	Air Force 1989b
Henry's law constant	1.00x10^{-4}–1.00x10^{1} atm-m^3/mol	Air Force 1989b
Autoignition temperature	246 °Cd	CRC 1984
Flashpoint	-23–1 °C	NFPA 1986
Flammability limits	1.3% lower; 8.0 upper	NFPA 1986
Explosive limits	No data	

[a] JP4, or jet propellant-4, is a mixed compound composed primarily of hydrocarbons (i.e., alkanes, cycloalkanes, alky-benzenes, indan/tetralins, and Naphthalenes).

[b] Jet fuels are blends prepared to meet certain gross property specifications. Most characteristic data only reflect gross properties covered in the specifications. Proportions and values vary with the type of crude oil from which the final fuel is derived and the refining process used.

APPENDIX E

Table E-4.a. Chemical Identity of Fuel Oils

Character	Information		
Chemical Name	Fuel oil No. 1[a]	Fuel Oil No. 2[b]	Fuel oil No. 6[c]
Synonym(s)	Kerosene, coal oil kerosine, range oil, straight run kerosene, distillate fuel oils light, furnace oil no. 1, Deobase®, JP-5, JP-1, range oil	API no. 2 fuel oil, gas oil, home heating oil no. 2, number 2 burner oil, diesel fuel, furnace oil no. 2	No. 6 fuel oil, Bunker C
Registered Trade Name(s)	Deobase®		
Identification Numbers:			
CAS Registry	8008-20-6	68476-30-2	68553-00-4
NIOSH RTECS	OA5500000	HZ1800000	LS8940000
EPA Hazardous Waste	No data	No data	No data
OHM/TADS	7217063	No data	No data
DOT/UN/NA/IMCO	UN 1223, IMO 3.3	No data	No data
shipping	632	No data	No data
HSDB	No data	C54795	No data
NCI			

[a] Fuel oil #1 is a mixture of C-9 through C-16 hydrocarbons primarily containing approximately 64% aliphatic hydrocarbons, 1–2% olefinic hydrocarbons, and 35% aromatic hydrocarbons.

[b] Fuel oil #2 is a mixture of C-11 to C-20 hydrocarbons.

[c] Fuel oil #6 is 25% aromatics, 15% paraffins, 45% naphthenes, and 15% non-hydrocarbon compounds

CAS = Chemical Abstracts Services; DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; EPA = Environmental Protection Agency; HSDB = Hazardous Substances Data Bank; NCI = National Cancer Institute NIOSH = National Institute for Occupational Safety and Health; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data Systems; RTECS = Registry of Toxic Effects of Chemical Substances

Source: ATSDR Toxicological Profile for Fuel Oils, 1995.

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Table E-4b. Analysis of Fuel Oils[a]

	Volume %		
Hydrocarbon type	Fuel oil no. 1	Fuel oil no. 2	Fuel Oil no. 6
Paraffins (*n*- and iso-)	52.4	41.3	5.9
Monocycloparaffins	21.3	22.1	3.9
Bicycloparaffins	5.1	9.6	3.4
Tricycloparaffins	0.8	2.3	2.9
Other cycloparaffins	-	-	5.0
Total saturated hydrocarbons	**79.7**	**75.3**	**21.1**
Olefins	No data	No data	No data
Alkylbenzenes	13.5	5.9	1.9
Indans/tetralins	3.3	4.1	2.1
Dinaphthenobenzenes/indenes	0.9	1.8	2.6
Naphthalenes	2.8	8.2	2.6
Biphenyls/acenaphthenes	0.4	2.6	3.1
Flurenes/acenphthylenes	No data	1.4	7.0
Phenanthrenes	No data	0.7	11.6
Other aromatic hydrocarbons	No data	No data	57.8
Total aromatic hydrocarbons	**23.6**	**24.7**	**78.9**

[a] Derived from IARC 1989; provided by the American Petroleum Institute

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Table E-4c. Physical and Chemical Properties of Fuel Oils

Property	Fuel oil no. 1	Fuel oil no. 2	Fuel oil no. 6
Molecular weight	No data	No data	No data
Color	Pale Yellow[b]; Colorless to brown[c,d]	Colorless to brown[c]	Colorless to brown
Physical state	Liquid[c]	Liquid[c]	Liquid
Melting point	-45.6 °C	-29 °C	No data
Boiling point	175–325 °C[b]; 200–260 °C[d]	160–360 °C[f]; 282–338 °C[d]	151-588 °C
Density at 15°C at 20 °C	 0.810–0.9360 g/mL[c] 0.80 g/mL[e,f]	 No data 0.8700–0.9500 [c]	 No data No data
Odor	Kerosene-like[c]	Kerosene-like[c]	Kerosene-like
Odor threshold (ppm)	0.082[f]; 1[d]	No data	No data
Solubility: Water at 20 °C Organic solvents	 ≈5 mg/L[c] Miscible with other petroleum solvents[b]	 ≈5 mg/L[c] No data	 ≈5 mg/L No data
Partition coefficients: Log K_{ow} Log K_{oc}	 3.3–7.06[c] 3.0–6.7[c]	 3.3–7.06[c] 3.0–6.7[c]	 3.3–7.06 3.0–6.7
Vapor pressure at 21 °C	2.12–26.4 mm Hg[c]	2.12–26.4 mm Hg[c]	No data
Henry's law constant at 20 °C - atm-m^3/mol	$5.9x10^{-5}$–7.4[c]	$5.9x10^{-5}$–7.4[c]	No data
Autoignition temperature	229 °C[d]	257 °C[d]	No data
Flashpoint (close cup)	38 °C[c,d]	58 °C[d]	No data
Flammability limits (% volume in air)	0.7–5%[d]	0.6–7.5%[d]	No data
Conversion factors	No data	No data	No data
Explosive limits	0.7–5%[b]	No data	No data

[a]Values listed are specifications required or general characteristics of each class of fuel oils
[b]HSDB 1991
[c]Air Force 1989
[d]Coast Guard 1985
[e]IARC 1989
Source: ATSDR toxicological profile for Fuel Oils

APPENDIX E

Table E-5.a. Chemical Identity of Mineral-Based Crankcase Oil

Character	Information	Reference
Chemical Name	Mineral-based crankcase oil	ATSDR 1997c
Synonym(s)	API 79-7; API service classification SAE 30 automotive motor oil; monograde automotive engine oil; multigrade automotive engine oil; marine engine oil; base engine oil; monograde diesel oil; railway diesel oil; marine diesel oil	ATSDR 1997c
Registered Trade Name(s)	Not applicable	
Chemical Formula	Not applicable	
Chemical Structure	Not applicable	
Identification Numbers:		
CAS Registry	8002-05-9	IARC 1984
NIOSH RTECS	No data	
EPA Hazardous Waste	No data	
OHM/TADS	No data	
DOT/UN/NA/IMCO shipping	No data	
HSDB	No data	
NCI	No data	

CAS = Chemical Abstracts Services; DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; EPA = Environmental Protection Agency; HSDB = Hazardous Substances Data Bank; NCI = National Cancer Institute
NIOSH = National Institute for Occupational Safety and Health; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data Systems; RTECS = Registry of Toxic Effects of Chemical Substances

Table E-5.b. Concentration of Components in Used Mineral-Based Crankcase Oil[a]

Component	Median concentration (ppm)
Arsenic	5
Barium	48
Cadmium	3
Chromium	6.5
Lead	240
Zinc	480
Dichlorodifluoromethane	20
Trichlorotrifluoroethane	160
1,1,1-Trichloroethane	200
Trichloroethylene	100
Tetrachloroethylene	106
Benzene	20
Toluene	380
Xylene	550
Benz(a)anthracene	12
Benzo(a)pyrene	10
Naphthalene	330
PCBs	5

[a] Environmental contamination from mineral-based crankcase oil is more likely to be from used crankcase oil than for the fresh products. Used oil is significantly contaminated with heavy metals and polycyclic aromatic hydrocarbons that are insignificant in the unused products. TPH results do not yield values for these contaminants, but the table is provided for informational purposes.

Source: ABB-Environmental Service, Inc. 1990

Table E-5.c. Physical and Chemical Properties of Mineral-Based Crankcase Oils

Property	Information	References
Molecular weight	No data	
Color	Yellow brown	DOE 1989
Physical state	Liquid, oily	DOE 1989
Melting point	-34.4 °C	DOE 1989
Boiling point	360 °C	DOE 1989
Density at 20 °C	Not applicable	
Odor	Lube oil odor	DOE 1989
Odor threshold (ppm)	No data	
Solubility		
Water at 20 °C	Insoluble	DOE 1989
Organic solvents	No data	
Partition coefficients:		
Log K_{ow}	No data	
Log K_{oc}	No data	
Vapor pressure at 21 °C	No data	
Henry's law constant at 20 °C - atm-m^3/mol	No data	
Autoignition temperature	≥135 °C	DOE 1989
Flashpoint (close cup)	≥163 °C	DOE 1989
Flammability limits (% volume in air)	No data	
Conversion factors	No data	
Explosive limits	No data	

Source: ATSDR. 1997. Toxicological profile for mineral-based crankcase oil. Agency for Toxic Substances and Disease Registry. Atlanta, GA.

APPENDIX E

Table E-6a. Chemical Identity of Mineral Oil[a]

Character	Information	Reference
Chemical Name	Mineral Oil	RTECS 1995
Synonym(s)	Paraffin oils, Heavy mineral oil, Light mineral oil, Liquid paraffin, Aliphatic petroleum hydrocarbons, Liquid Vaseline, Paraffins, Paroleine, Liquid Petrolatum, White Mineral Oil, White Oils	RTECS 1995; ATSDR 1997b
Registered Trade Name(s)	Nujol, Thermia C, ADEPSINE OIL, ALBOLINE, Balneol, BAYOL F, Bayol 55, Blandlube, Crystosol, Drakeol, FLEXON 845, Fonoline, GLYMOL, Crystosol, IRGAWAX 361, KAYDOL, Kondremul, MagieSol 44, Molol, Neo-Cultol, Parol, Peneteck, Penreco, Perfecta, Petrogalar, PRIMOL D, Primol 355, Protopet, SAXOL, SHELLFLEX 371N, SUNPAR 150, Tech Pet F, ULTROL 7, UVASOL	
Identification Numbers:		
CAS Registry	8020-83-5; 8012-95-1	RTECS 1995
NIOSH RTECS	LX3300000	RTECS 1995
EPA Hazardous Waste	No data	
OHM/TADS	7217073	OHM/TADS 1991
DOT/UN/NA/IMCO shipping	UN1203, UN1257	RTECS 1995
HSDB	No data	
NCI	No data	

[a] Mineral oil refers to classes of petroleum hydrocarbons whose origin is petroleum distillation streams. Light paraffinic (naphthenic) distillate contains C_{15}–C_{30} hydrocarbons; heavy paraffinic (naphthenic) distillate contains C_{20}–C_{50} hydrocarbons; white mineral oil contains C_{15}–C_{50} hydrocarbons, and petrolatum and most residual oils contains $>C_{25}$ hydrocarbons. Source: IARC 1984.

CAS = Chemical Abstracts Services; DOT/UN/NA/IMCO = Department of Transportation/United Nations/North America/International Maritime Dangerous Goods Code; EPA = Environmental Protection Agency; HSDB = Hazardous Substances Data Bank; NCI = National Cancer Institute; NIOSH = National Institute for Occupational Safety and Health; OHM/TADS = Oil and Hazardous Materials/Technical Assistance Data Systems; RTECS = Registry of Toxic Effects of Chemical Substances

APPENDIX E

Table E-6.b. Physical and Chemical Properties of Mineral Oil[a]

Property	Information	Reference
Molecular weight	No data	
Color	Clear	HSDB 1998
Physical state	Liquid	HSDB 1998
Melting point	No data	
Boiling point	360°C	HSDB 1998
Density at 20/4 °C	0.875–0.905	HSDB 1998
Odor	Like burned lubricating oil	HSDB 1998
Odor threshold: Water Air	No data	
Solubility: Water at 25 °C Organic solvents	 Insoluble Soluble	 HSDB 1998 HSDB 1998
Partition coefficients: Log K_{ow} Log K_{oc}	 No data No data	
Vapor pressure at 20/30 °C	No data	
Henry's law constant at 24.8 °C	No data	
Autoignition temperature	500–700°F	HSDB 1998
Flashpoint	135°C (closed cup) 193°C (open cup)	HSDB 1998 HSDB 1998
Flammability limits in air	No data	
Conversion factors: ppm (v/v) to mg/m^3 in air at 25 °C	 1 ppm (v/v) =3.96 mg/m^3	
mg/m^3 to ppm (v/v) in air at 25 °C	1 mg/m^3 =0.25 ppm (v/v)	
Explosive limits	No data	

[a] Mineral oil refers to classes of petroleum hydrocarbons whose origin is petroleum distillation streams. Light paraffinic (naphthenic) distillate contains C_{15}–C_{30} hydrocarbons; heavy paraffinic (naphthenic) distillate contains C_{20}–C_{50} hydrocarbons; white mineral oil contains C_{50} hydrocarbons, and; petrolatum and most residual oils contains >C_{25} hydrocarbons. Source: IARC 1984.

Source: HSDB. 1998. Hazardous Substances Database. Environmental Protection Agency, available through National Library of Medicine, MEDLARS, Washington, DC.

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